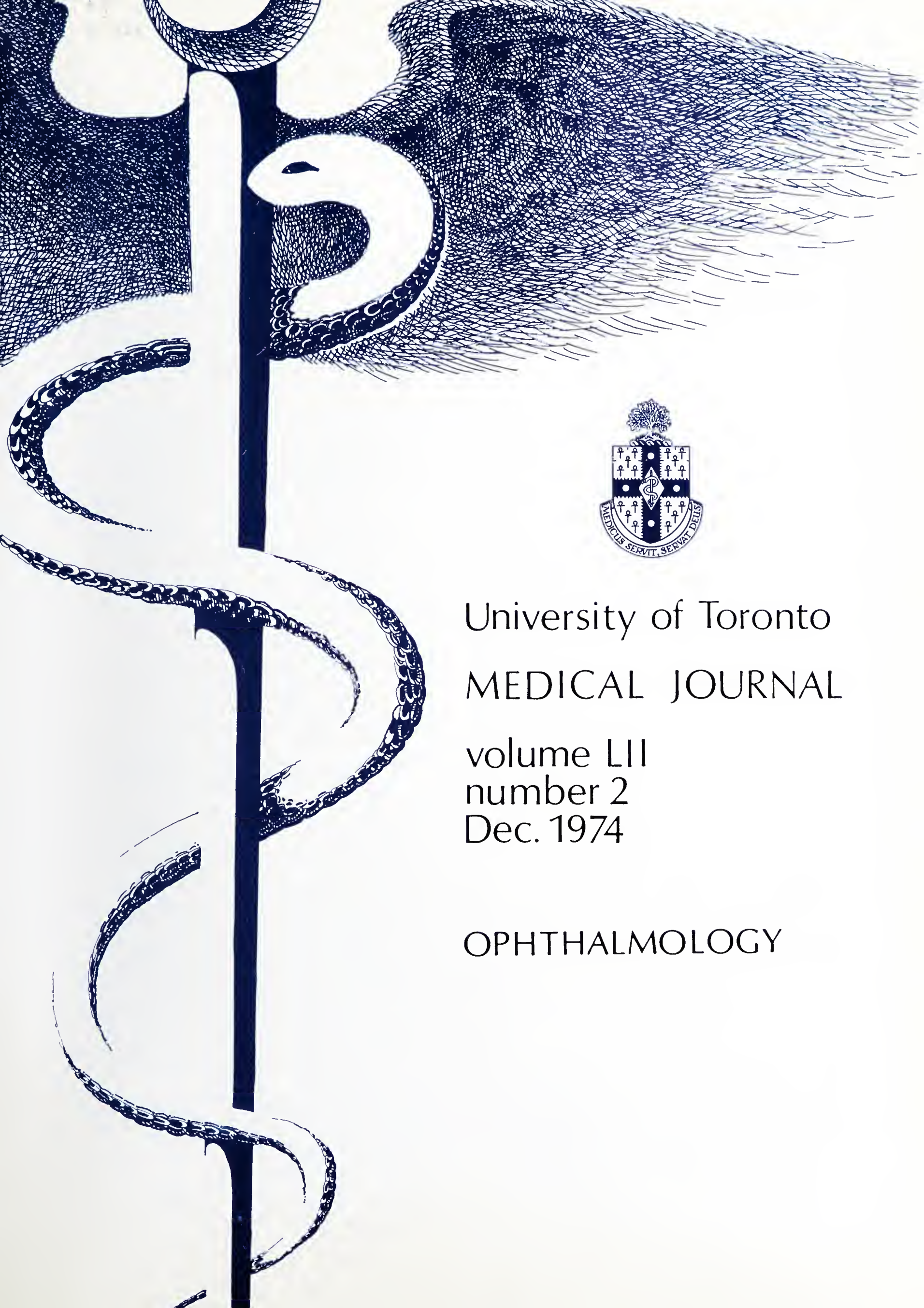

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Guest Editorial

Clement McCulloch received his M.D. at the University of Toronto Medical School in 1939. He took his ophthalmology training at the Institute of Ophthalmology at the Columbia University Medical School in New York. He was then in the medical section of the Royal Canadian Air Force.

After the war Dr. McCulloch was on staff at the Toronto Western Hospital until he became head of the Department of Ophthalmology of the University of Toronto in 1961. In the same year he was appointed as ophthalmologist-in-chief at the Toronto General Hospital.

Dr. McCulloch has published over 50 scientific papers and is internationally known for his work on choroideremia and the zonule of the lens. He has been instrumental in the establishment of the ophthalmic research laboratory located at 1 Spadina Crescent.

Good Vision? Well, who hasn't?

J. C. McCulloch, M.D.

In our complex western society good vision is taken for granted. But, what is good vision? What we really are talking about is superlative vision. Each of us expects to go about the demanding, technical, visually exact tasks of our civilized culture with no visual difficulty, in fact, with ocular comfort and without distress. It is truly remarkable that the large proportion of people have, or are assisted to have, perfect vision, and hold this throughout their working life and well into old age.

What is known as 20/20 vision is an astonishing achievement. Look at a "20" letter on a visual acuity chart next time you pass an eye clinic. It is amazingly little, yet it must be read at 20 feet. But this task is structured on the components of the letter, which are 1/5 of its size, and are ridiculously small. And this must be projected to the retina, which by geometry then becomes a tiny distance, just one cell across at this location. At the receptor cell the physical components of the light involved are at their limits of smallness. Note also that the optical surfaces and media of the eye have minimal aberrations, that the system of pickup has a power magnification worthy of astronomy and that the neurological connections have a sophistication which will teach lessons to our computer friends for generations to come. All of this each of us expects as a birthright, assumes will remain with us all our life, without weariness or let up.

Ophthalmology is a small specialty in the big field of medicine, and it is only right that it be so, for the eye is but a little organ. Yet it is paramount. Marshall McLuhan's comment that "The media is the message," certainly applies to the eye. Man consists of a brain with an eye stuck in front of it. The rest of his body is assembled to nourish, assist and carry about those important organs. The neurologic connections of the eye are the largest single component of the nervous system. The cells set aside to look after vision are more than those for all the other four senses combined. The eye feeds the brain with information. The brain not only uses this at the moment it is received, but as we develop throughout childhood the brain organizes all its complexity of responses from input supplied in large part by the eye. A person born without the use of eyes has not the opportunity to develop from lack of sight.

What are the tools used to look after civilized man's eyes? Firstly there are the tools of optics. The tolerances achieved in optical components are among the crowning accomplishments of civilized man. The succession of Newton rings displayed in the Russian Pavilion at Expo in Montreal were quietly reproduced in another form in the German Pavilion – two nations using this example to tell us of their technical excellence. Modern spectacles are mass-produced with rigid quality control. Lenses may be clear, tinted or variable in tint; they may be reflection free. Different glass can be fused into combined lenses, without flaw and with great precision. The testing procedures to decide on the glass to be used are exact and reproduceable. The millions of people wearing glasses attest to the efficiency of the industry and the accuracy of those wedding the patient to the lens.

If you don't like glasses, you can have contact lenses. These rapidly are reaching the tolerances achieved by regular glasses, are more becoming, and allow wider, more natural vision. There are intraocular lenses, that can be put in at operation for cataract; these are not new, many thousands have been used. They are still being developed but one can't doubt that they will find a place as a modality of treatment.

To go over to other tools used to treat people's eyes, there is surgery. If the cornea becomes opaque it can be replaced by a transplant. If the lens is the offender a cataract extraction is done and the lens is replaced by glasses, contacts or an intraocular lens. If the retina tears and is detached the tear is

sealed and the retina reattached. If a retinal vessel bleeds, or becomes incomplete leading to local edema, or blocks with local infarct as in diabetes, the area can be dried up with a laser. A child's eye may be straightened and then the child taught to reassemble in his brain correctly placed images. The surrounding structures of the eye, lids, tear passages, orbit and neighbouring face may all be repaired or reassembled to protect or subserve the eye.

There are still more tools in ophthalmology. Obvious ones are the medical ones. Many systemic diseases display themselves in the eye. Treatment for the eye may be the general treatment that is necessary for the whole person. Hypertensive retinopathy clears amazingly with medical control. Diabetic retinopathy is harder to sort out but certainly good control of the diabetes is good treatment for the ocular complications. The list of medical disorders involving the eyes leads to neurological diseases. Here the relationship is intimate, complex, and in its very complexity stimulating. We are only beginning to understand how the eye movements are controlled, but what we do know is one of the great sagas of modern medicine. Similarly, how the brain succeeds in seeing and relating that to the sensory, motor, and affective total of the individual is just in the early stages of discovery.

There are other tools of ophthalmology, in fact many. One is an attribute that marks the outstanding practitioner from the ordinary. It is an appreciation of the social aspects of ophthalmic practice and of the human being in his setting. Successful translation of sound medical or surgical procedures into a rehabilitated, satisfied, functioning and effective patient may finally hinge on that warm humane quality of a good physician. This is particularly so in ophthalmology, where a threat to that gem of an organ, the eye, leads to the greatest of emotion in the person. Success is not the well done operation but the person again using his sight.

One cannot see other than that our civilization will become more complex, the eye more important, and the care of the eye more exact, more extensive and more rewarding.

Recycled Eyeglasses give new Sight to Latin Americans

Under the joint sponsorship of the Christian Medical Society and Evangelical Medical Missionary Aid Society (E.M.M.A.S.), the sixth "Eye" Medical Group Mission in the Dominican Republic was conducted from November 9th to 23rd.

The team, including twenty-eight Ontario residents and some twenty-five Americans, was composed of ophthalmologists and one ophthalmic resident, optometrists, opticians, nurses and para-medical personnel. They worked in a remote area on the northeast coast of the Dominican Republic where little eye care was available to these needy people. Accompanying the team was approximately one ton of used eyeglasses collected by Service Clubs, Church groups, school children, etc., new glasses donated by Canadian optical houses and department stores, and medical and surgical supplies donated by Ontario medical and surgical supply houses.

Most of the personnel pay their own airfare and accommodation but a few are subsidized by E.M.M.A.S.

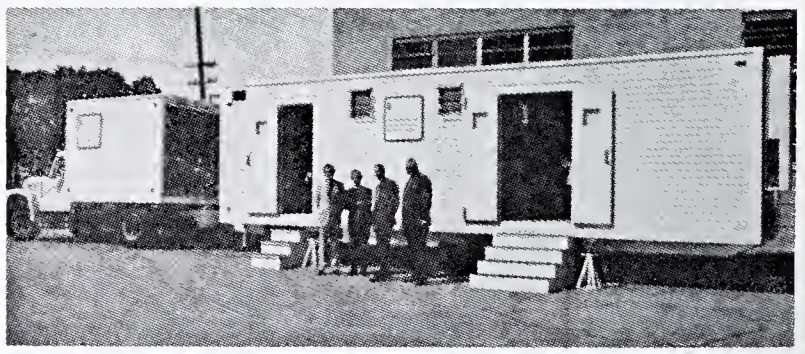
Approximately six thousand patients were seen in consultation, and of these some four thousand received eyeglasses to correct their presbyopia and myopia. Most of these patients had never had a pair of glasses.

Using the combined facilities of our mobile operating van and the small new hospital, one hundred and eleven ophthalmic surgical procedures were performed. Over half of these were cataract operations performed on patients led into clinic blind. The majority of the cataract operations were on an out-patient basis. Before departure each cataract patient was given a pair of "cataract temporary glasses".

These eye missions are only a part of the total program of general medical, surgical and dental missions carried on in the Republic of Honduras, Haiti, Dominican Republic and Liberia (West Africa).

Further eye programs are planned for 1975.

D. W. HARPER, M.D.
Medical Group Missions Representative
Evangelical Medical Missionary Aid Society



Pictured above is the mobile surgical unit used in the Eye programs. The truck contains a diesel generator which powers the X-ray machine and operating room equipment within the trailer.

Neuro-Ophthalmology: The Branch with the Neurological Twist

*J. Raymond Buncic, M.D., F.R.C.S.(C)**

Neuro-ophthalmology is the study of the neurology of the visual system, and, in its broadest sense, deals with how we perceive our world. In the sum total of all the modes in which the body "feels" its environment, a huge share of our brain function is concerned with the most economic way of *seeing* our surroundings. Let us take, for example, the visual activity of a medical student in the library. He walks into a brightly lit room, seeing the library before him, and his *pupils constrict* to monitor the appropriate amount of light entering his eyeballs. With the voluntary *rapid* (saccadic) movement of his eyes, he *fixates* with both retinal *maculae* in *parallel* fashion on a certain book in the bookshelf across the room. He *maintains his fixation* on the book as he walks toward it, seeing the people around him out of the corner of his eye, his *visual field,* and as he approaches the stacks, his eyes gradually *converge* as they reach a point from which he can read the title. As he reads the title, his eyes have converged, his pupils become smaller, and the power of his lenses has increased to keep the print in sharp focus on his retina *(accommodation)*. Suddenly, he notices some movement to his right in his peripheral field and with a rapid turn, his eyes move under *frontal lobe* control to his new object of regard, a friend in the distance who waves as he walks by *(refixation)*. His eyes are now rediverged in parallel and move *slowly* from midline to the right under *occipital control* as he follows his friend across the room with his eyes. There is a sudden breeze from the open window, and his eyes *blink* rapidly several times in response to the new *sensation* on the cornea, to protect it from harmful stimulation and keep it covered with a comfortable layer of tear film.

The ultimate aim of this neurological economy is to achieve an accurate visual appreciation of our environment. Not only is a high fidelity receiving system or afferent system necessary for this (optical media, retina, optic nerve and visual pathways to occipital cortices), but also an oculomotor or efferent system is obligatory to maintain the eyes stable on one object, whether still or moving, so that true binocular single vision and depth perception or stereopsis are possible. In other words, the oculomotor system must be able to maintain the eyes in a quiet position as they gaze on an object, maintain them parallel as they follow an object in the distance which moves across their visual field, and converge the eyes as the object approaches and rediverge as the object recedes. With the central and most sensitive points of our retina, the maculae, we are able to see minute objects with maximum acuity, but, at the same time, can perceive the visual context with the more peripheral portion of the retina and its subservient visual pathway. When some aspect of the visual perception system breaks down, be it the afferent portion or the efferent, we appreciate a visual disturbance such as blurred vision, diplopia, darkness or scintillating scotomata. It is the task of neuro-ophthalmology to examine the economy of this system in order to detect and treat or help in the management of the disorders discovered. With this intention in mind, we can systematically examine the afferent and efferent visual systems, sometimes with special ophthalmological gadgetry, with most attention paid to those areas suggested by the symptomatology.

THE AFFERENT VISUAL SYSTEM

Most complaints of visual loss, such as dimming or blurring of vision, blackness in some area of vision, or cloudiness, are due to some interruption of the afferent visual stimuli. Occasionally these symptoms may be due to an inability to co-ordinate the eyes or the inability to move them in certain directions because of a paresis, but let us consider the problems associated solely with an inefficient afferent system. Under normal circumstances the vision in each eye is symmetrical and a bright light shone into one eye constricts the pupil directly and the partner pupil consensually to an equal degree. Also, the colour appreciation (retinal cone function) in each eye is the same. Take the case of a young man who has suddenly experienced visual loss in one eye which he refers to as a large gray patch in front of his eye associated with some pain on movement of that eye; we pay particular attention on our examination to that receptor apparatus. By physical examination, in our patient we find the ocular media to be entirely clear of opacities and the retina entirely normal in appearance. The optic nerve head or papilla also appears normal. A measurement of the visual acuity in that eye shows it is reduced below the normal 20/20 to 20/400's. This is not correctable by holding a pin-hole in front of his eye (the pin hole obviates any refractive difficulty and gives a true picture of the organic intactness as far as vision is concerned). Shining a bright light into the affected eye constricts the pupils only moderately, both directly and consensually. Bringing the light into the normal eye, however, causes brisk and greater constriction of both pupils. Swinging the flashlight from eye to eye demonstrates that as the light approaches the 'poor' eye the pupils dilate, and as the light approaches the 'good' eye both pupils con-

* *Staff Ophthalmologist, Hospital for Sick Children, Toronto, Ontario*

strict, and so as the light swings back and forth the pupils dilate and constrict in alternating fashion. This demonstrates clinically the so-called *'afferent pupillary defect'* or *'Marcus-Gunn pupil'*. Testing his colour vision either grossly by having him compare the brightness of a red pencil held before each eye or by more sophisticated colour vision tests brings to his attention that he can see red only as gray in the affected eye (dyschromatopsia). The visual field examination by simple confrontation technique using a pencil or a pinhead, by tangent screen examination using various size test objects under standard conditions, or by other forms of standard perimetry demonstrates a large central blind spot or 'scotoma' in the affected eye, but a normal field in the other eye. All our tests so far have been normal in the asymptomatic eye. Having the history and now the physical findings, we conclude from our knowledge of the natural history of disease that our patient is suffering from retrobulbar neuritis, which occasionally is an early presentation of multiple sclerosis. In cases in which both optic nerves would be similarly involved, the asymmetry of both the complaints and the physical findings would not be so great, and signs would be pathological in both eyes. Since we know that the optic nerves join to form the chiasm in which the medial retinal and optic nerve fibres decussate to the contralateral cerebral hemisphere, the visual field examination gives the clue to the localization of the defect in the afferent system. For example, the finding of a bitemporal hemianopia is pathognomonic of a lesion involving the optic chiasm.

Crucial information can be obtained from intelligent use of the ophthalmoscope. Visualization of the ocular fundus allows direct observation and assessment of its various elements and layers – the retina and its arteriovenous system, the choroidal (vascular) layer and the optic nerve head. Pathologic processes can often easily be seen – evidence of retinal ischemia as in carotid occlusive disease or chronic anemia, retinal arteriovenous occlusions, edema and detachments of the retina, choroidal metastases, and evidence of systemic diseases such as hypertension, diabetes mellitus and hyperviscosity states. Also important is the direct diagnosis of raised intracranial pressure, acute or chronic, by visible edema of the optic nerve head, i.e. papilloedema.

It sometimes happens that a patient complains of poor vision "on the right"; here we must distinguish between failure of afferent input to the right eye or to the right visual field consequent to a right homonymous hemianopia. How is vision when the left eye is covered? How is vision when the right eye is covered? These are questions which must be asked to help the patient make this distinction in his symptoms. Because of the decussation of the fibres in the chiasm, it turns out that the left hemisphere, as for other body functions, "controls" the contralateral visual function. Similarly, the right hemisphere "controls" the left visual field. For this reason, unilateral cerebral lesions cause various patterns of loss of field to one side almost equally in both eyes (homonymous hemianopia). Knowledge of the characteristics of a field defect permits the examiner to deduce to within reasonable limits the site of the cerebral lesion. Temporal lobe tumours, for instance, tend to cause contralateral upper hemianopic defects which enlarge as the tumour grows, whereas parietal lobe lesions tend to cause more inferior hemianopic field defects. Because the visual radiations are quite diffusely scattered as they course posteriorly in the temporal and parietal lobes, a certain degree of asymmetry (incongruity) is to be expected in the visual field defects caused by pathology in these areas. In the occipital lobe, however, the optic radiations have become concentrated in organized and orderly fashion so that small defects here produce perfectly symmetrical loss in homonymous fields of each eye.

In summary, defects of afferent visual flow are uncovered by the history of the symptoms and the examination of the functions which depend on either central or peripheral vision. In other words, the visual acuity, colour vision and visual fields are all affected to various degrees. A more indirect sign, which does not depend on any answer from the patient, is the comparison of pupillary responses to light between the two eyes, or simply noting if the pupillary response to light is abnormally diminished in the affected eye or eyes. Ophthalmoscopy may reveal directly pathology of the most anterior portion of the visual apparatus (globe, optic nerve, sometimes chiasm), but at other times visual field defects, oculomotor problems and accompanying neurologic signs may be more valuable in the diagnosis.

THE EFFERENT SYSTEM

Normally our eyes are still as we fixate on an object, and we are able to maintain this stable position of our eyes during fixation. Sometimes the stability of the eyes is upset and spontaneous pathological eye movements override our volitional attempt to maintain fixation. Sometimes also our eyes do not act in coordinated fashion; one eye may move to look in the new direction while the other lags behind, e.g. extraocular muscle palsies. Sometimes we are unable to look in one of the usual directions of gaze with our either eye because of a 'gaze' palsy. In general terms, symptoms of blurred vision, poor vision, or double vision (diplopia), are the clues to an oculomotor abnormality. We should, therefore, be able to examine the oculomotor system in a systematic way in order to explain the symptomatology.

The control of eye movements represents a complex neurologic organization, including both volitional systems and reflex systems[3, 4, 5, 6]. From the cerebral

level, frontal and occipital cortical outflow descends through the midbrain, to the pons and then to the three cranial nuclei (the third, fourth and sixth) and nerves, and finally to the extraocular muscles which control the movement of the globes. This pathway from above is also influenced by an inhibitory and controlling cerebellar input as well as by the vestibular apparatus, including the vestibular labyrinths. The types of oculomotor problems which can arise from some upset in this complex neurologic control is limited, but not completely understood. Spontaneous eye movements may appear which make it impossible for the eyes to rest in a stable position, such as 'nystagmus' or 'opsoclonus'. Palsies may exist due to lesions at any level of the motor control system, from cerebral problems to extraocular muscle paralysis, and each level of difficulty gives rise to its own characteristic form of eye movement disorder. Generally, lesions above the level of the three oculomotor nuclei produce a directional or gaze palsy of the eyes, termed a 'supranuclear' palsy because of its etiologic site. Characteristic of the supranuclear palsy is the ability to move both eyes in a given direction, the absence of any strabismus when the eyes are observed, and the absence of any diplopia. Because the eyes continue to remain parallel, they can both observe the same visual object and hence no diplopia is appreciated. If the lesion is at the nuclear level, however, the eye movement palsy is asymmetrical, depending on which cranial nerve nuclei are involved; diplopia becomes a problem. In a sixth nerve palsy, the affected eye can no longer abduct and is pulled unresistingly inward toward the nose, producing a crossed eye appearance (esotropia). The images seen by the two eyes will now be no longer appreciated as one but seen as separate in the horizontal plane. When the connection between the third, fourth and sixth cranial nuclei, the medial longitudinal fasiculus, is disrupted, as it may be by a small vascular lesion or demyelinating plaque, a disconnection syndrome results. Normally intactness of the medial longtitudinal fasiculus (MLF) is necessary to co-ordinate horizontal movements to either side. With lesions in this area, the medial rectus is disconnected from an attempt, either reflex or volitional, to gaze to the opposite side, while the abducting eye makes the move and sometimes enters a nystagmus phase. This constellation of eye signs, accompanied by complaints of blurring of vision and occasionally diplopia, is classic and referred to as 'internuclear ophthalmoplegia', typical of a vascular lesion if unilateral, and of multiple sclerosis if bilateral. In summary, palsies may be supranuclear, nuclear, internuclear and infranuclear. Primitive ocular reflexes help distinguish certain levels of motor involvement in the examination of the neuro-ophthalmologic patient. In the oculocephalic manoeuver, the head is turned from side to side fairly rapidly, and as the head is turned to one side the eyes move toward the opposite side; this is a useful reflex test in the comatose patient. It demonstrates the intactness of the vestibular apparatus and its brainstem connection with the MLF and the pons, which contains the horizontal gaze mechanism responsible for the conjugate eye movements. The same vestibulo-ocular pathway can be examined with the use of caloric stimulation using either warm or cold water to irrigate the external ear canal, a cold water 'stimulation' usually producing a tonic deviation toward the irrigated ear in a comatose patient, and vestibular nystagmus towards the contralateral ear in a conscious patient. A cranial nerve palsy clearly would prevent the movement of an eye normally, when the oculocephalic manoeuver or calorics are used. For this reason, it is valuable to remember that in supranuclear palsies, these two brainstem tests, as well as Bell's phenomenon, are usually normal.

One can look at the supranuclear palsies in an even more refined way: the frontopontine pathways are responsible for quick volitional or saccadic movements, and this differentiates them from the occipitopontine pathway, which is responsible for the involuntary smooth pursuit type of eye movement. It is possible to get affection, for instance, of the frontal portion of this mechanism with preservation of the occipital portion. In such a case as this, e.g. the Steele-Richardson Syndrome, the patient is able to follow a fixated object while it moves in his field of vision, but he has lost the capacity to make voluntary rapid eye movements (saccades). Here again, because of the supranuclear nature of the lesion, brain stem reflexes are intact, with reservation, however, that the rapid movements which form the fast or jerk part of the vestibular nystagmus are absent during testing with calorics in the conscious patient.

Besides ocular motor paresis, a very common type of disorder is the presence of pathological spontaneous eye movements of an incoordinated fashion. Nystagmus is one of the most common of these varieties, and may be described as repetitive involuntary oscillations of the eyes. Nystagmus is usually bilateral and symmetrical, but it may be highly asymmetric or even monocular. There are many varieties of nystagmus and the mechanism for the production of the various forms is not well understood. Like all disorders, it can be classified as congenital or acquired. There are also some physiologic forms of nystagmus which can be elicited clinically, such as vestibular nystagmus seen after rotation in a chair or irrigation of the ear with warm or cold water, or optokinetic nystagmus. Congenital nystagmus is often pendular but may be jerky, and is usually characterized by a history of presence since birth or early childhood, and sometimes a family history. Acquired nystagmus is usually hallmarked by the history, acquired symptomatology, or a more neurological context. Vertical nystagmus is usually pathological and may be drug induced, such as by tranquilizers and barbiturates. Otherwise, vertical nystagmus, and often nystagmus evoked by gazing to the right or left, so-called gaze nystagmus, are usually

associated with brainstem disorders. Convergence-retraction nystagmus is typical for pathology in the area of the midbrain such as pinealoma. The dissociated nystagmus seen in the abducting eye of internuclear ophthalmoplegia is also characteristic for the MLF lesion. A more rare variety of nystagmus called 'see-saw' nystagmus is seen with large perisellar lesions such as large craniopharyngiomas, pituitary adenomas or gliomas in that area. A very coarse gaze nystagmus may represent partial recovery from a gaze paresis in which attempts to maintain gaze to one side are made but cannot be sustained but for short periods of time. Good short reviews of these oculomotor problems are available[1, 4]. More complex oculomotor disorders such as opsoclonus, ocular dysmetria and flutter oscillations as seen in cerebellar disease, and ocular bobbing as found in catastrophic pontine haemorrhages, are well reviewed in the larger reference texts[1, 3].

Besides being an excellent clue for the existence of afferent and visual defects, an understanding of the normal pupillary morphology and response to light and to convergence is important in the avoidance of confusion with other more serious neurological conditions. The parasympathetic nerve supply to the eye constricts the pupil and is also involved in tear secretion. It is carried to the eye by the lower division of the third nerve and then to the pupillary sphincter and ciliary muscle for accommodation. For this reason pupillary dilatation is often the earliest manifestation of a third nerve palsy, and may be a clue to uncal herniation in a comatose patient or the existence of a ruptured intracranial ancursym in a patient who has developed a sudden headache and stiff neck. The 'tonic pupil', usually a large, slowly reacting pupil whose pathology resides in the ciliary ganglion, may be mistaken for an early third nerve palsy with its dilated pupil. A small pupil which dilates poorly in the dark and is associated with some ptosis represents a Horner's Syndrome involving the sympathetic nerve supply to the eye. This may be the only clue to an apical lung carcinoma affecting the paravertebral autonomic ganglia at the base of the neck. The lids are influenced by both the third nerve and the seventh cranial nerves. During a blink, the levator of the lid supplied by the third nerve relaxes while the orbicularis oculi supplied by the seventh nerve contracts. At the end of the blink the levator is reactivated and the lid rises. With a third nerve palsy, the lid droops; with a seventh nerve palsy, however, the orbicularis oculi supplies no resistance to the pull of the levator and the lid is slightly wider than usual. The blink is the motor part of the protective mechanism of the eye along with the exquisite corneal sensitivity supplied by the ophthalmic division of the trigeminal nerve.

The neuro-ophthalmologist attempts by history and physical examination to pinpoint the level of visual difficulty, be it afferent or efferent. He attempts to localize problems of the retina, optic nerve, chiasm, optic tract or visual radiations by using his special techniques of physical examination. He relies heavily on the use of his knowledge of the corrected visual acuity, field examination, pupillary responses, and ophthalmoscopy to locate a lesion of the retino-occipital pathways. In the efferent or motor system, close attention is paid to the ability of the eyes to fixate on small objects, the presence of a strabismus, examination for ocular palsics, of rapid and slow eye movements, the observation of abnormal spontaneous movements such as nystagmus, and the examination of brain stem oculomotor reflexes. To supplement the physical examination special investigative techniques are available such as fluorescein angiography for the examination of blood flow through the choroid and retina and the demonstration of retinal vascular and optic nerve problems; electroretinograms (ERG), to substantiate the presence of retinal dystrophy such as retinitis pigmentosa with visual loss; and visual evoked responses (VER), for the person with cerebral blindness or malingering.

Knowledge of the functional anatomy of the brain is necessary to enable us to correlate the neuro-ophthalmologic signs into meaningful patterns which result in in accurate localization anatomically of the neurologic lesion. For example, difficulty with a blink, absent corneal reflex, and esotropia due to sixth nerve palsy, all on the same side, in association with a mild hearing loss ipsilaterally, together suggest a cerebellopontine angle lesion such as an acoustic neuroma. One must weigh all the evidence in the context of the general neurological picture when the final judgement is made as to site and type of lesion affecting the oculomotor and visual systems. Because of the variety of diseases producing neuro-ophthalmologic syndromes, it is natural and mandatory for the neuro-ophthalmologist to work hand in hand with the complementary specialties such as neurosurgery, neurology and internal medicine, towards complete diagnosis and care of the patient.

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The Essentials of Glaucoma

J. Donald Morin, M.D.
Norman S. Hill, 7T6

WHAT IS GLAUCOMA?

Glaucoma is the single most frequent cause of blindness in people over 40 years of age. It is a common disease, slowly progressive, and is often initially asymptomatic. Most importantly, glaucoma is treatable so that blindness can usually be avoided. A few cases present acutely, but these also can be treated before damage to the eye is irreversible. Early diagnosis is required to prevent visual loss. It is therefore important that every physician, especially the general practitioner and internist, be aware of the initial signs of glaucoma, and of the population which is at risk, in order that treatment may be started quickly.

Glaucoma is a group of diseases which is characterized by increased intraocular pressure due to an obstruction to the outflow of aqueous from the eyeball. Aqueous humor is produced by the ciliary body and secreted into the posterior chamber. Then it flows between the lens and the iris through the pupil into the anterior chamber. Aqueous drains out of the anterior chamber through the trabecular meshwork into the canal of Schlemm, and finally empties into the venous system. Glaucoma is usually due to an increased outflow resistance and is rarely the result of increased aqueous production. This common type is known as *primary open angle glaucoma* or *chronic simple glaucoma.* If the trabecular meshwork is obstructed by the peripheral iris because of a narrow angle between the iris and the cornea, *acute angle closure* develops.

WHOM DOES GLAUCOMA AFFECT?

A common disease in most parts of the world, the incidence of glaucoma has been estimated to be 2% of the population over 40 years of age. Younger people do develop glaucoma in which case it may be masked as progressive myopia. The high frequency of this disease is suggestive of a Mendelian dominant type of transmission, and indeed, this was believed to be true for many years. It is now thought that glaucoma is transmitted as an autosomal recessive trait which is found in a large segment of the population. The incidence of glaucoma occurring in a second family member is 15 to 20%. Therefore, a family history of glaucoma and examination of the relatives of an afflicted patient are very important in the early detection of this disease. Certain other disorders are known statistically to be associated with a higher incidence of glaucoma. Diabetes mellitus, cataract, myopia, central vein occlusion, topical steroids and eye injuries are common examples. The alert physician will be watching for evidence of glaucoma in these high risk patients.

WHAT ARE THE PRESENTING SYMPTOMS?

It is unfortunate that the typical onset of glaucoma is insidious and asymptomatic until visual impairment occurs, because by this time it may be too late to salvage useful vision. There are no symptoms that are pathognomonic of glaucoma but the diagnosis should be suspected if the patient presents with blurred vision, mild headache, impaired dark adaptation, a sensation of halos around electric light bulbs or has required frequent lens changes. The headaches may be unilateral since one eye is affected earlier than the other. Visual field changes, as measured on a tangent screen, are diagnosic of glaucoma and provide the best method of following the course of the disease; however, changes which are subjective to the patient are indicative of very advanced disease. The measurement of the intraocular pressure is the easiest technique available to the general practitioner and internist to diagnose glaucoma.

About 10% of primary glaucoma is *acute narrow angle glaucoma* due to a sudden closure of the angle between the cornea and the iris. The acute attack is characterized by a sudden onset of blurred or foggy vision, usually unilateral, followed by severe, sometimes throbbing pain localized around the eye and over the distribution of the sensory division of the fifth cranial nerve. Nausea and vomiting, often accompanying the acute episode, are frequently diagnosed as an

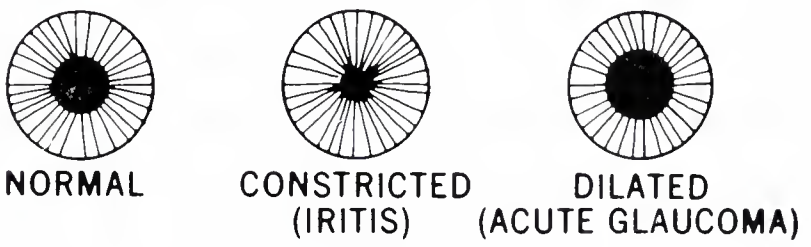


FIGURE 1

acute abdominal condition. Physical findings include edema of the cornea, redness of the bulbar conjunctiva, a fixed moderately dilated pupil, (fig. 1) and elevated intraocular pressure. Repeated attacks, as well as prolonged attacks, will eventually lead to permanent visual defects. Both must receive immediate and surgical attention.

The pain associated with *acute angle glaucoma* is commonly over the distribution of V_1 and V_2. It is necessary, then, for the physician to include tension headache, migraine headache, frontal neuritis, temporal arteritis and nasal sinusitis in the differential diagnosis. Two other eye disorders associated with ocular pain of sudden onset are acute arteritis and acute conjunctivitis. The patient with acute iritis has subjectively less pain, normal intraocular pressure, a constricted pupil,

moderately decreased vision and a clear cornea. Acute conjunctivitis presents with pain of a burning nature, no visual loss, a mucopurulent eye discharge, intensely inflamed conjunctiva, normal pupillary responses, a clear cornea and normal intraocular pressure.

Cases of *secondary glaucoma* account for between 20 and 40% of all cases of raised intraocular pressure and usually occur following intraocular hemorrhage due to trauma or surgical procedures on the eyes, or as a complication of other eye disease such as uveitis, cataract or tumours. The clinical onset may be acute or insidious, but the physician should be watching for signs that glaucoma may be complicating any primary eye disorder.

HOW IS THE DIAGNOSIS MADE?

Measurement of the intraocular pressure is the most definitive indicator of glaucoma. A Schiotz tonometer, used to measure the intraocular pressure, is an inexpensive piece of equipment which is easy to use and of great value in accurately diagnosing glaucoma (fig. 2). If a patient presents with symptoms of glaucoma, it is likely that irreversible damage has already occurred. In order to catch the disease early and before damage has been done, measurement of intraocular pressure should be a routine part of the physical examination of every patient over 40 years of age. The following is an account of the procedure used.

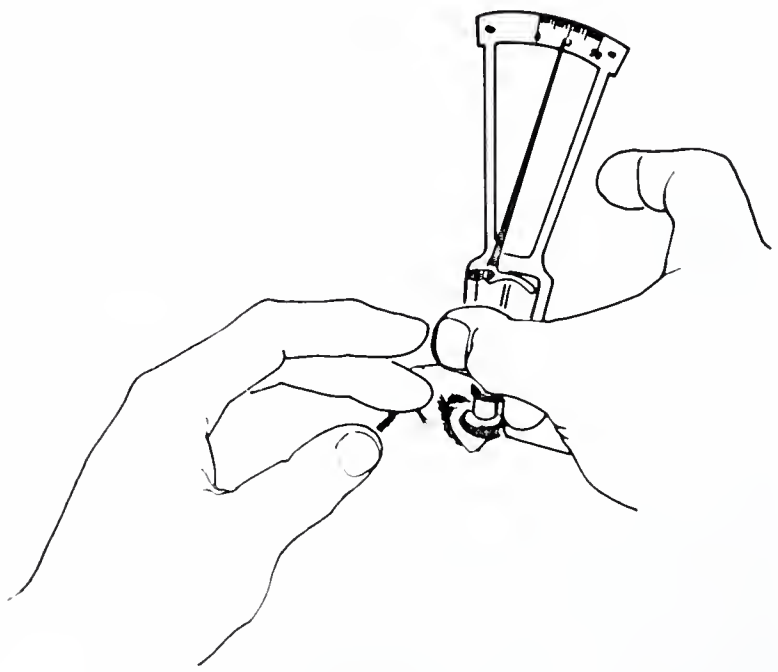


FIGURE 2

With the patient in the supine position the cornea is desensitized by applying a topical anesthetic such as 0.5% tetracaine. When the anesthetic has taken effect, the patient is asked to stare at the ceiling while the tonometer is placed perfectly vertical on each cornea, and a scale reading taken. Charts accompanying the tonometer convert this reading into millimetres Hg of intraocular pressure. Between 12 and 20 mm Hg, intraocular pressure is considered normal. A difference between the two eyes of greater than 3 mm Hg is unusual in the absence of disease. Any pressure above 20 mm Hg may damage the optic nerve, depending on the individual susceptibility of the patient. A reading of 20 to 25 mm Hg is highly suggestive of glaucoma and this patient should be referred for more extensive investigation. Referral is particularly important if this patient has not yet developed symptoms or if he has a family history of glaucoma. The technical simplicity and the potential reward of early glaucoma diagnosis are the benefits of tonometry and cannot be overemphasized.

Optic atrophy occurs with retrobulbar neuritis, syphilis, central retinal artery occlusion, trauma and any lesion damaging the optic nerve along its intracanalicular or intracranial course. Glaucoma is also associated with optic atrophy and causes characteristic disc changes which tend to occur as the disease progresses. These changes indicate visual impairment has begun.

The region of the optic disc receives its blood supply from small branches of the ophthalmic artery. It is generally held that the increased intraocular pressure of glaucoma compresses these vessels, leaving the disc ischemic and that this prolonged ischemia leads to the optic disc changes and neural tissue destruction. The earliest visible change is a slight increase in the physiologic cupping of the disc. As the process continues, the cup becomes increasingly wide, deep and vertically oval. The temporal or neuroretinal rim of the disc takes on a light grey or white hue and becomes notched. The retinal vessels are displaced nasally (fig. 3).

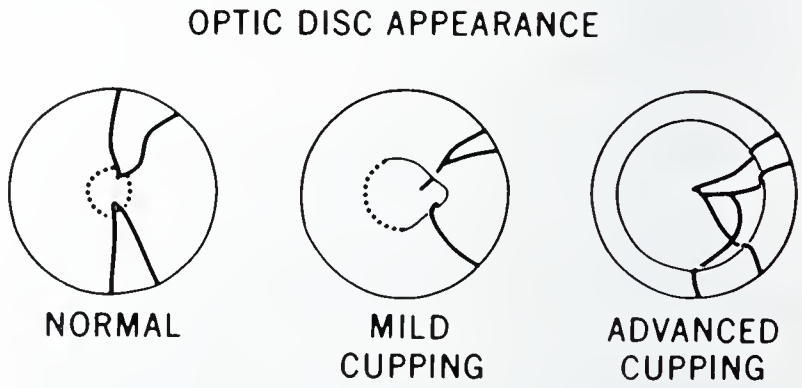


FIGURE 3

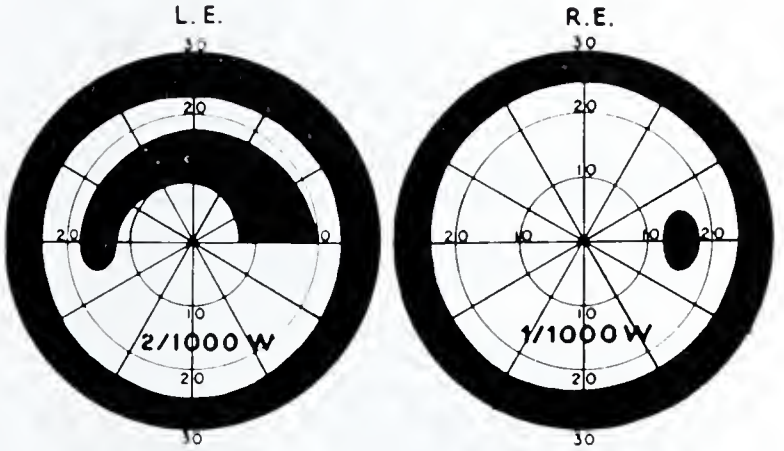
These changes are not invariably due to glaucoma but this is the first diagnosis until proven otherwise. Findings such as raised intraocular pressure may be used to confirm the diagnosis of glaucoma. The appearance of the disc gives an estimate of the prognosis. For example, an optic disc which is not markedly cupped will not likely be associated with severe neural or visual damage.

Ideally, the diagnosis of glaucoma is made before visual field changes become subjective to the patient. If the patient is aware of his decreased vision, then much destruction has already occurred. Mapping of the

visual fields on a tangent screen provides an excellent measure of the severity of the disease. It is the best way to evaluate the effectiveness of treatment. Although damage to the neural tissue is irreversible, medical treatment can usually halt enlargement of scotomata. The visual field changes are due to retinal nerve fibre bundles being damaged as they pass into the optic nerve at the disc. The nasal and superior fields are typically the first to be lost with peripheral field loss occuring later. Most often the last remaining area of vision is a small, inferior, temporal island (fig. 4). Some patients may be left with relatively good central vision for a long time before blindness finally occurs.

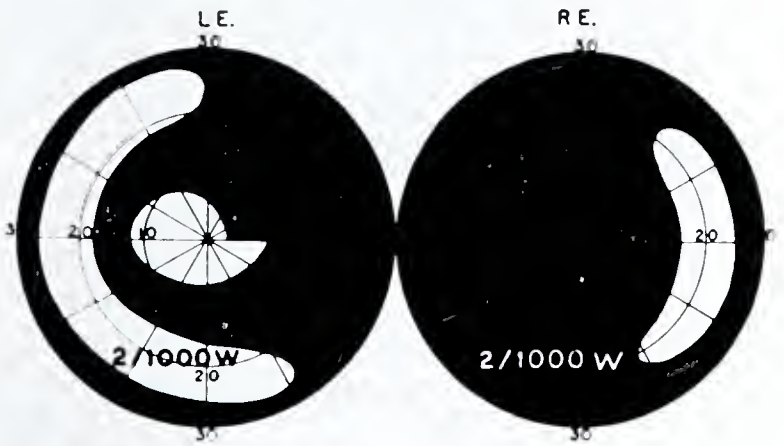
HOW IS GLAUCOMA TREATED?

The approach to the treatment of glaucoma varies, depending on whether the disease is *chronic open angle* or *acute narrow angle*. If it occurs secondary to another ocular disorder, the underlying condition must be corrected first. Whether primary or secondary in nature, the principle of treatment is to reduce the inflow or assist the outflow of the aqueous humor and thereby lower intraocular tension.



2) Upper Nerve Fiber Bundle Defect

1) Enlargement of Blind Spot



3) Double Nerve Fiber Bundle Defect with Nasal Step

4) Residual Field

FIGURE 4

The majority of cases are adequately controlled by topical application of a miotic agent. Miotic eyedrops open the angle between the iris and the cornea in narrow angle eyes, allowing aqueous to flow out through the canal of Schlemm. In open angle eyes, miotics exert tension on the ciliary muscle and hence on the trabecular meshwork, thereby opening up its passageways. Two commonly used miotics are pilocarpine and carbachol which may be used up to 3 to 4 times per day. The weakest drug concentration should be started initially, increasing the dosage gradually until the effective concentration is reached. This approach will minimize the side effects such as headache, which are transient and usually disappear spontaneously after a week of treatment. In the meantime, the side effects are controlled by salicylates.

If miotics alone do not lower the intraocular pressure, a carbonic anhydrase inhibitor may be added to reduce aqueous secretion. Carbonic anhydrase is an enzyme required to produce normal amounts of aqueous. Therefore, blocking this enzyme reduces aqueous production. The most frequently used preparations are acetazolamide, 125 to 250 mg q.i.d., and dichlorphenamide, 25 to 100 mg per day. While prolonged treatment with these drugs occasionally causes renal calculi and other side effects, their use in elderly or debilitated people may prevent the necessity of surgery.

Surgical intervention is delayed as long as possible in chronic glaucoma because such procedures tend to hasten the onset of cataract formation. Therefore, surgery is not considered unless the intraocular pressure cannot be maintained below 25 mm Hg by medication or if loss of visual fields continues to progress. If surgery is indicated, aqueous formation can be decreased by destroying part of the ciliary body, or a drainage canal can be formed between the anterior chamber and the subconjunctival space to increase aqueous outflow.

An attack of *acute glaucoma* is both a medical and surgical emergency. Vigorous medical treatment to lower the intraocular pressure is always attempted first. A regimen of oral glycerine, 1 to 2 mg/kg body weight in 50% solution or other hyperosmotic agents, 500 to 1000 mg acetazolamide and 4% pilocarpine, 2 drops every 15 minutes is used to lower the pressure. This combination of drugs almost always succeeds in reducing the intraocular pressure, thereby stopping the acute attack. However, between 20 and 40% of patients have repeated attacks of angle closure despite prophylactic miotic therapy, so curative surgery is recommended. The operation used in this instance is the resection of part of the peripheral iris (peripheral iridectomy). New outflow tracts may have to be constructed if peripheral iridectomy fails to lower the pressure. Although *acute onset glaucoma* usually presents unilaterally, it is a bilateral disease and in time the other eye will become involved. In this situation it is advisable that peripheral iridectomy be performed before the inevitable acute attack occurs.

The gift of vision is one of life's luxuries that is difficult to fully appreciate until it is lost. Since the days of the Greek empire, glaucoma has been a major cause of impaired vision and blindness. Now, with advances in medicine, much of the anguish brought on by glaucoma can be avoided. Glaucoma, as a prevalent genetic weakness, cannot be eliminated from our society, but with prompt diagnosis and careful medical management, it can cease to be such a devastating disorder.

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Book Review

GENERAL OPHTHALMOLOGY

EDITORS: Daniel Vaughan and Taylor Asbury
Lange Medical Publishers, Los Altos, 1974 (7th edition), 334p. Price: $9.50.

Vaughan's *General Ophthalmology*, 7th edition, has arrived at last! It remains the most practical text available for period three ophthalmology. Changes in format make this new edition easier to scan. Particularly welcome is a chapter on the use of the direct ophthalmoscope. There is also a section on tears (not the student's); this is most relevant since the dry eye is the most common problem one will treat in the elderly. An understanding of the disturbed physiology allows one to approach therapy rationally. The chapter on drugs has been expanded and ultrasound examination has been introduced into the discussion of vitreous lesions. Otherwise, the text is unchanged. This very readable volume will provide the interested student with a complete survey of the extent and depth of eye disorders.

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Herpes Zoster Ophthalmicus - New Perspectives

INTRODUCTION

Herpes zoster (shingles) has been recognized as a specific clinical entity since the time of the ancient Greeks, when it was called zona (meaning girdle) because of its characteristic bandlike distribution around the trunk.

Zoster, which normally occurs sporadically and infrequently in the general population, assumes new significance because the growing number of old, debilitated and immunosuppressed patients has led to an increase in its incidence. Herpes zoster ophthalmicus is especially important because of the relatively common association with severe post-herpetic neuralgia and ocular complications resulting in permanent eye damage, including blindness.

ETIOLOGY

Herpes zoster occurs as a result of infection with the varicella zoster (V-Z) virus, a member of the herpes virus group. It is an icosahedral DNA virus, 204 by 240 nm, composed of 162 capsomeres and surrounded by a lipid envelope. The V-Z virus is morphologically identical to the herpes simplex virus and the cutaneous lesions of varicella, h. zoster and h. simplex infection are all histologically similar.

There are two current theories on herpes zoster infection. The first is that it may result from exposure of a partially immune individual to the V-Z virus (this is the so-called *epidemic* or *idiopathic herpes zoster*). In other words, first exposure to the virus (usually in childhood) results in a primary immunological response and the clinical manifestation of varicella (chickenpox). Subsequent exposure to the virus leads to an accelerated, secondary immunologic response and the clinical manifestation of herpes zoster.

The second theory is that infection represents activitation of a latent virus present in dorsal root nerves and cranial nerve ganglia (the so-called *symptomatic herpes zoster*). In this case, primary infection allows some viruses to enter nerve cells remaining dormant until some time later in life when activation is caused by non-specific stress (such as exposure to cold, pressure on a nerve root, arsenic, tuberculosis, or a fresh massive dose of V-Z virus).

The close association between varicella and herpes zoster is based on several factors, including the identity of the causative virus, similar histopathology, cross agglutination and complement fixation reactions, and clinical resemblance. In fact, it may be that chickenpox represents the dermotropic manifestations of the V-Z virus and herpes zoster the neurotropic properties.

EPIDEMIOLOGY

Herpes zoster is a sporadic disease with world-wide distribution, showing no seasonal preference, and occurs more commonly in males. It is a disease which rarely occurs before the age of twenty (although cases have been reported in children as young as 3½ years old[5]), two thirds of the cases being in patients over 45 years. The incidence of herpes zoster has been reported as 2 per 1,000 people in a large city[13]. The most common site of involvement is the thorax and trunk (from the second dorsal to the second lumbar nerves), representing about 75-80% of all cases. Herpes zoster ophthalmicus is the next most common, representing about 15% of all cases[13]. It is most commonly the ophthalmic division of the trigeminal nerve that is affected (see figure 1), but on rare occasions there may be involvement of the maxillary or mandibular divisions.

FIGURE 1

Patient with a typical case of herpes zoster ophthalmicus. The lesions are distributed unilaterally (with sharp demarcation in the midline of the forehead), and predominantly over the ophthalmic division of the fifth cranial nerve. Note the prominent swelling of both upper and lower lids and the vesicular eruptions over the lateral forehead and temporal regions. Some vesicles have ruptured leaving an overlying crust or dark ulcerated lesions. There does not appear to be corneal involvement.

The communicability of herpes zoster does not seem to be great. It is infectious for the first two or three days following the appearance of eruptions, and during this time it may initiate an outbreak of varicella in susceptible children. It rarely causes zoster in other adults. By way of contrast, varicella is an extremely infectious disease among children, and it may cause herpes zoster in exposed adults.

A case of varicella confers lasting immunity to reinfection, but only partial immunity to herpes zoster. Herpes zoster also confers lasting immunity to reinfection. Cases of varicella and zoster occurring simultaneously in a child have been reported[5], but these are usually rare.

Factors predisposing to herpes zoster infection are: systemic malignancy (eg. Hodgkin's disease), previous radiation therapy, impaired cellular immunity, prior splenectomy and chemotherapy with immunosuppressive drugs.

PATHOPHYSIOLOGY

Herpes zoster ophthalmicus may be described pathologically as an inflammation of the gasserian ganglion and certain sensory nerve roots of the ophthalmic division of the trigeminal nerve. It is characterized by vesicular eruptions in those areas of skin supplied by the affected nerves.

The virus has a "colchicine-like" cytopathogenic effect on human cells, causing arrest in metaphase, overcontracted chromosomes, chromosome breaks and often formation of micronuclei.

Microscopically, the affected ganglion shows lymphocytic infiltration, leptomeningitis and neuronal necrosis. It is also possible to demonstrate eosinophilic intranuclear inclusion bodies which, in the process of nuclear degeneration, may be seen to enter the cytoplasm. The inflammatory process most often extends along the frontal nerve of V_1, quite often sparing the nasociliary and lacrimal branches. Spread to the cornea occurs through the ciliary branches of the nasociliary nerve. The inflammatory process may also involve other ganglia (eg. ciliary and geniculate) and nerve fiber sheaths.

In mild cases there is little or no permanent, damage, while in more severe cases there may be scarring in the involved ganglion.

In the early stages of epidermal involvement there is some proliferation of the epidermis, and the spheroidal eosinophilic inclusion bodies may be seen in the infected cells. As in neuronal involvement, these inclusion bodies may be seen to enter the cytoplasm after nuclear degeneration. Later stages show amitoses of the prickle cell layer, formation of (sickle-shaped) multinuclear cells, "ballooning degeneration", loss of reticulation, final disintegration of cells, and accumulation of tissue fluid resulting in the typical vesicles. The dermis is also affected in the necrotic process, as evidenced by the deeply pitted scars left after healing.

In complicated cases of herpes zoster ophthalmicus the same sorts of inflammatory processes may manifest as conjunctivitis, scleritis, uveitis, optic neuritis and keratitis.

CLINICAL FEATURES

(i) *Manifestations*: The incubation period of herpes zoster ophthalmicus seems to be about 7-14 days. The onset is sudden and unexpected, ushered in by fever, malaise, and considerable neuralgia and tenderness along the affected dermatome. There may also be general discomfort, headache, nausea and vomiting.

Three or four days later there is blushing and an increase in temperature of the skin in the involved area, which is invariably unilateral, giving the clinical picture of an erythematous dermatitis. This is followed by edema and then crops of papules progressing to vesicles on an erythematous base. Initially the vesicles contain a clear fluid but this rapidly becomes purulent. Within a short time these vesicles burst, forming ulcers which may become secondarily infected with bacteria. The pain usually disappears with the formation of yellowish crust-like scabs which eventually separate, revealing the pitted scars. Recovery in uncomplicated cases is complete in 3-6 weeks for adults and 2-3 weeks for children.

Herpes zoster in the absence of typical vesiculation *("herpes zoster sine herpete")* may be a more common variant than previously suspected[8]. Rarely, the typical vesiculation is replaced by a seborrhoeic cutaneous lesion.

Hutchinson's rule states that if there is involvement of the side and tip of the nose, there will be involvement of the cornea. This follows logically from the local anatomy, as both the side of the nose and the cornea are supplied by the nasociliary nerve. Although a good general rule, it is not invariably true, and Schwartz[14] has reported one case of a young girl with nasociliary involvement and no corneal lesions.

Pritikin and Duchon[3], in a study of 2,300 cases of herpes zoster ophthalmicus, reported corneal involvement in about 40%. It begins with diffuse edema and diminished sensation. This is followed by a punctate epithelial keratitis, which soon develops into a combined epithelial and subepithelial keratitis and then into the nummular type of stromal opacity (see figure 2). Over the stromal lesions epithelial vesiculation may occur, going on to exfoliation and ulceration. These ulcers are very susceptible to infection, difficult to treat, and slow to heal. The punctate infiltrate may settle in 2 to 3 weeks or may drag on, unresponsive to treatment, for months.

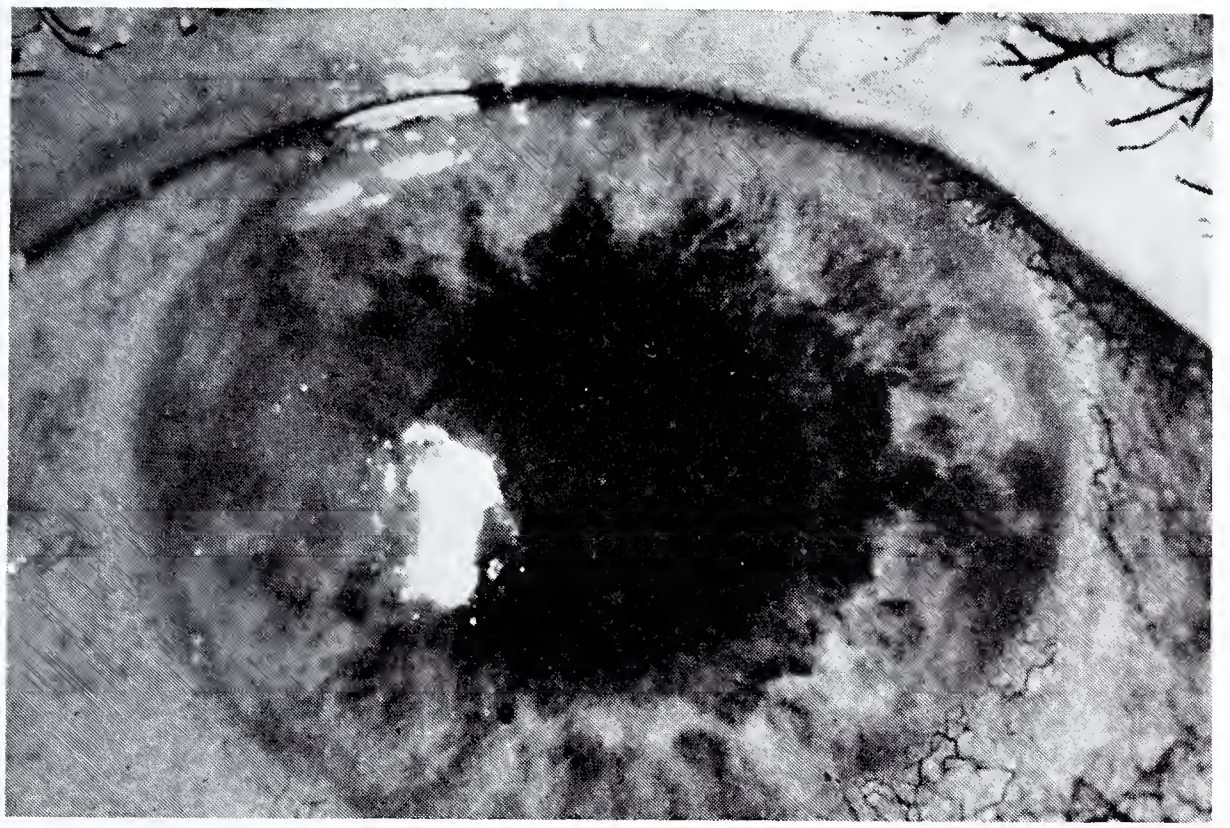


FIGURE 2

Herpes zoster keratitis. Note the irregular light reflex indictating corneal ulceration, and the numerous, punctate stromal opacities. There is also considerable injection of the conjunctival vessels. The pupil appears regular and there is no evidence of synechiae.

Examination of the eye during the eruptive period is made difficult by edema of the upper lid, vesiculation and hyperalgesia. Conjunctival lesions are infrequent, but when present are found in conjunction with injection of the mucosa and occasionally petechial hemorrhages.

In some cases, after the appearance of the localized lesion, the patient may go on to develop *"herpes zoster generalisata"* (disseminated zoster) which is a generalized vesicular eruption simulating varicella. In a study by Stevens and Merigan[10], there was an association between disseminated zoster and concurrent disease, especially Hodgkin's disease.

Local confinement of the disease and recovery seem to depend on the patient's ability to mobilize a prompt systemic, anamnestic humoral response, as well as a local response in the primary dermatome. Especially important are (1) the appearance of virus-specific complement fixing antibody, and (2) local interferon production. Clinical improvement is usually seen to follow 48 hours after peak interferon levels.

(ii) *Laboratory Diagnosis*: A smear of vesicular fluid 24 to 48 hours after the appearance of the lesions stained with Giemsa shows many multinucleated giant cells and type A inclusion bodies. Cerebrospinal fluid may be abnormal, showing increased pressure and a lymphocytic pleocytosis.

The diagnosis may be made retrospectively by demonstrating a rise in antibodies to vesicular fluid in convalescent serum. It is also possible to demonstrate specific antibodies in the patient's serum by the immunofluorescence technique. Complement fixing antigen is also found within the vesicles.

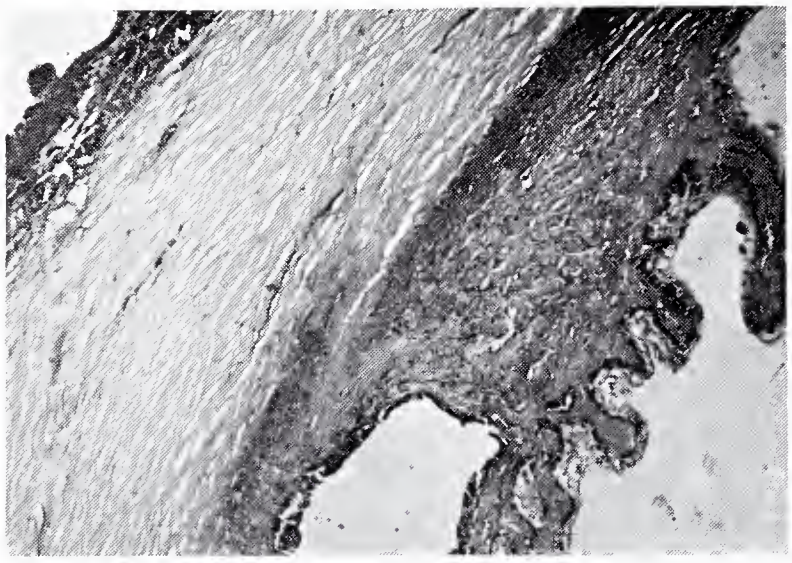
It is important to note that the V-Z virus has a cytopathogenic effect in human tissue culture only, and cannot infect other animals (such as chick embryos, rabbits and mice used commonly in laboratory diagnosis).

(iii) *Differential Diagnosis*· Herpes zoster ophthalmicus often poses a diagnostic problem in the preeruptive stages because the pain is non-specific and may simulate other more common conditions.

In the eruptive stage herpes zoster ophthalmicus is most often confused with herpes simplex, varicella, or dermatitis herpetiformis. The distinction from varicella and D. H. is usually easily made on the basis of the clinical picture, history and age of the patient. Herpes simplex may offer more of a problem, especially if it is one of the rare cases which is confined unilaterally to a single nerve distribution. If there is some doubt, the distinction may be made on the basis of the ability of vesicular fluid from one of the lesions to infect the scarified cornea of a rabbit (h. simplex can infect the rabbit, but V-Z virus cannot). If the patient has corneal ulceration, the lesion of h. simplex will characteristically be dendritic, whereas that of h. zoster is usually a non-specific superficial keratitis, and on occasion, a disciform keratitis.

(iv) *Complications*: Some of the more common complications of herpes zoster ophthalmicus are listed below, but it is important to note that both the frequency and severity of complications increase with increasing age of the patient. In addition, older patients are more likely to heal lesions with scarring.

Ocular complications are most common, occurring in about 50% of all cases of herpes zoster ophthalmicus. The most common of these is keratitis which is often accompanied by uveitis with contraction of the pupil and photophobia. In severe corneal involve-

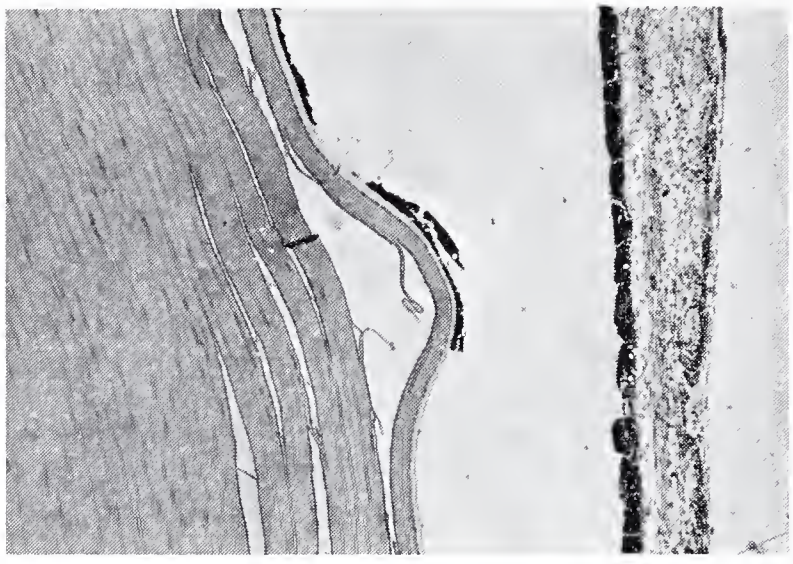


FIGURES 3 (ABOVE) AND 4 (BELOW)

Photomicrographs of the enucleated eye of a 79 year old woman, illustrating some of the complications of herpes zoster ophthalmicus. Infection in this case was followed by an iritis and secondary glaucoma. There was progressive visual deterioration to complete blindness, and the ocular pressure at the time of enucleation was 80 mm Hg.

In figure 3 a portion of the corneal epithelium can be seen in the extreme upper left hand corner. Immediately below this and overlying the corneal stroma are markedly congested limbal vessels. There is a chronic inflammatory cell infiltrate in this region and in the ciliary body seen to the right and middle of the photograph. Fibrosis of the ciliary body and processes is also evident.

Figure 4 shows a portion of the anterior lens and iris. There are numerous chronic inflammatory cells in the iris stroma and evidence of a ruptured synechia in the centre of the photograph.



ment there is an increased chance of developing (1) neuroparalytic keratitis, (2) severe iridocyclitis, (3) scleritis, and (4) as a much later sequela, secondary glaucoma (see figures 3 and 4).

Less common ocular complications include a paralytic mydriasis or Argyll-Robertson pupil due to ciliary ganglion involvement and iritis. Herpetic optic neuritis is a rare complication which follows an acute course lasting a few weeks, and has an uncertain prognosis for the recovery of vision.

The next most common complication is post-herpetic neuralgia. This may be very severe and incapacitating, occurring in about 30% of patients over 40 with herpes zoster ophthalmicus[13]. If the affected area is left with diminished sensitivity in combination with persistant post-herpetic neuralgia, the patient is said to have *"anesthesia dolorosa"*.

Roughly 13% of all cases of herpes zoster ophthalmicus will be left with a temporary ocular palsy[13]. This is due to involvement of one or all of the third, fourth and sixth cranial nerves[12].

A rare but none-the-less serious complication is meningoencephalitis.

Hematogenous dissemination of the virus and systemic involvement is a rare complication. However, this can be a potentially lethal situation in a compromised host who is unable to produce sufficient levels of antibody and interferon to halt the infection. A patient who presents initially with herpes zoster ophthalmicus and who goes on to develop disseminated zoster should always be suspected of having a malignancy.

TREATMENT

The early neuralgic manifestations of herpes zoster ophthalmicus are best managed with sedation, rest and mild analgesics such as aspirin, although more potent agents such as codeine might be necessary in the severe cases.

Systemic corticosteroids started early in the course of the disease may be helpful in reducing the incidence and severity of post-herpetic neuralgia and ocular complications. (Typical daily dosages are 40 mg. of prednisone or 4 to 6 mg of dexamethasone.)

The skin lesions in the earliest erythematovesicular stage can be treated with a shake lotion of alcohol and menthol or camphor. In the early vesicular stage, painting the lesions with equal parts of compound tincture of benzoin and flexible collodin gives effective, comforting relief. In the vesiculopustular stage systemic and topical antibiotics are useful in preventing secondary bacterial infection.

An antibiotic ointment in combination with a topical corticosteroid is especially effective in the treatment of corneal lesions. In all cases of keratitis it is important to keep the pupil dilated with atropine 1% or cyclopentolate 0.5 to 1%, 1 drop t.i.d. to prevent formation of synechiae (adhesions of the posterior iris to the lens). It is also important to monitor intraocular pressure in the convalescing patients because of the relatively common incidence of secondary glaucoma. In serious cases, or where permanent damage is feared, ophthalmic consultation is advised.

Post-herpetic pain is difficult to treat and it has a poor prognosis if it is persistent or if it occurs in the elderly. On occasion this pain may be severe enough to cause marked depression, and cases of suicide have been reported. In these patients powerful analgesics of the morphine group may be justified. Other measures,

including deep X-ray therapy, pituitrin and vitamin B_1, sodium iodide i.v., ganglionectomy or section of the nerve root are usually ineffective. Epstein, in the J.A.M.A. (1969)[9], reported good results in the treatment of post-herpetic pain using intralesional, subcutaneous injections of 200 mg triamcinalone diacetate diluted with 100 ml 2% procaine hydrochloride.

In immunosuppressed patients or patients with immunodeficiency, gamma globulin prepared from pooled plasma of patients convalescing from herpes zoster may be helpful as a means of post-exposure prophylaxis. The dose is 20 ml intramuscularly daily for 4 to 7 days. Cytosine arabinoside in massive i.v. doses for 2 days has been reported to prevent further dissemination of lesions within 48 hours, in cases of *"herpes zoster generalisata"*.

PROGNOSIS

Recovery is uneventful in normal, healthy individuals. The prognosis for permanent eye damage and especially post-herpetic pain worsens with increasing age of the patient.

SUMMARY

This paper deals with some of the more important aspects of herpes zoster ophthalmicus. Its cause, clinical aspects, and current methods of treatment have been reviewed with special reference to the more common complications which arise during or shortly after the disease. The increased vulnerability of old, debilitated and immunosuppressed patients to this infection has been emphasized.

ACKNOWLEDGEMENTS

I would like to express my appreciation to all those who helped prepare and review this paper. My special thanks to Dr. J. B. Walter of the Department of Pathology for his excellent clinical photograph, and also Barbara Moreau, Eileen Girard and Frank Carré of the Department of Ophthalmology for their help with the slides and photography.

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Lasers in Ophthalmology

Rod Underell, 7T6

The first operational laser was built in 1960 at Hughes Aircraft Research Laboratories by T. H. Maiman. Since that time, "laser" has become almost a household word. In addition to the popular developments in laser technology, the physical and biological sciences have put lasers to immense use in a wide variety of research. Of all fields in medicine to benefit thus far from laser technology, ophthalmology has gained the most. This article attempts to explain the theory of lasers including a description of the models of lasers used in ophthalmology and their clinical application. Some possible future trends for laser technology in the practice of medicine will also be discussed.

THE THEORY OF LASER OPERATION[1]

As most people probably know, the word "laser" is abbreviated from the first letters of the words *L*ight *A*mplification by the *S*timulated *E*mission of *R*adiation. The first two words explain the laser is an amplifier and the signals it amplifies are light signals or photons. The rest of the words, "by the Stimulated Emission of Radiation", tell one how this amplification is accomplished.

Any electron in an atom can only exist in that atom at certain discrete levels of energy. An electron's energy is not continuously variable, but variable only between several particular values. These particular values are known as energy levels. The lowest particular energy level is called the ground state and all other

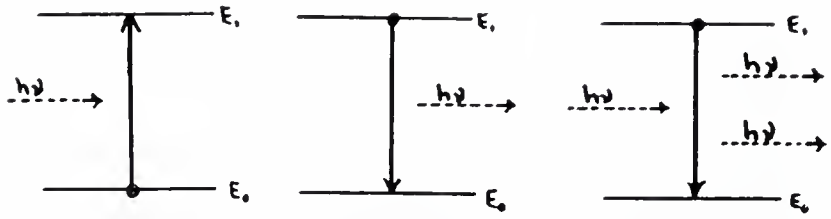
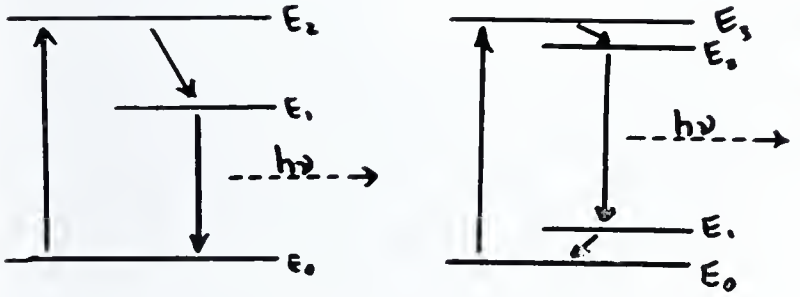


FIGURE 1

energy levels are called excited states. In Figure 1, the three possible ways an electron transfers from one energy level to another are shown for an electron with only two possible energy levels, E_0, the ground state, and E_1, the sole excited state. The three possible methods of energy transfer are absorption, spontaneous emission and the one of interest for lasers, stimulated emission. Recalling Planck's equation $E=h\nu$ where E is photon energy, h is Planck's Constant and ν is the frequency of the radiation, absorption of energy by an atom and the movement of an electron from E_0 to E_1 will occur when an incoming photon with energy $h\nu=E_1-E_0$ provides the exact quantized amount. Similarly, once an electron is in E_1, it may decay spontaneously back to E_0, emitting as it does so a photon of energy $E=E_1-E_0$. However, suppose instead, that before the electron spontaneously decays, a second photon with appropriate energy is absorbed by the atom. The electron still falls back to E_0, but now *two* photons are emitted in the process. Moreover, these photons are identical in frequency and highly coherent. The initial photon has been amplified into two.

In most situations stimulated emission is most unlikely to happen. In a large system of atoms with the two energy levels shown, the majority of atoms are in the energy state E_0. (To be precise, the quantized Boltzmann distribution for this population in normal thermal equilibrium gives $N_1=N_0 \exp\left(\frac{E_1-E_0}{kT}\right)$ where N_1 is the number of particles in E_1, N_0 is the number of particles in E_0, k is Boltzmann's Constant and T is the Absolute Temperature.) Thus a photon with appropriate frequency entering the population or system is more likely to strike an atom in the ground state than an atom in the excited state. The photon is more likely to be absorbed than to undergo stimulated emission. Suppose then that the photon does strike an atom in E_0 and excites the electron to E_1. The next incoming photon will have an increased probability of striking an excited atom, but only on the condition that the next photon arrives in the population before one of the subgroup of excited atoms spontaneously decays. The life time of an atom in the excited state (E_1) prior to decay can be measured and is characterized by the symbol τ. From this simple model, two conclusions may be arrived at. One is that light amplification by stimulated emission requires that high numbers of atoms in a potential lasing population be in excited states. (For example, in our model the condition is $N_1>N_0$. At $N_1=N_0$, fifty percent of incoming photons (assuming no spontaneous emission over this time) will be absorbed while the other fifty percent will be doubled to give again the original intensity. With $N_1<N_0$, the intensity of the beam will be reduced in passing through this population.) The second conclusion is that photon input must be extremely rapid if the τ is short. Thus, in trying to construct a laser, it would be most convenient to choose a population of atoms with as long a τ as possible. The terminology used to describe these excitation events is *pumping* an active laser media to achieve *population inversion*. This can be translated to mean supplying appropriate frequency energy quickly enough to a population of atoms with a τ long enough, in order that the normal energy distribution of many atoms with little energy and few with a lot has been "inverted" to many in high levels and fewer in the ground state. Once a photon is introduced into this population a chain reaction of decay is started, one into two, two into four, and so on, each link providing output photons nearly identical to each

other and to the input photon; consequently, any given photon is nearly identical to the original photon and to all others being produced in the population. It is this nearly perfect reproduction that gives laser light its unique properties.



Three Level System Four Level System

FIGURE 2

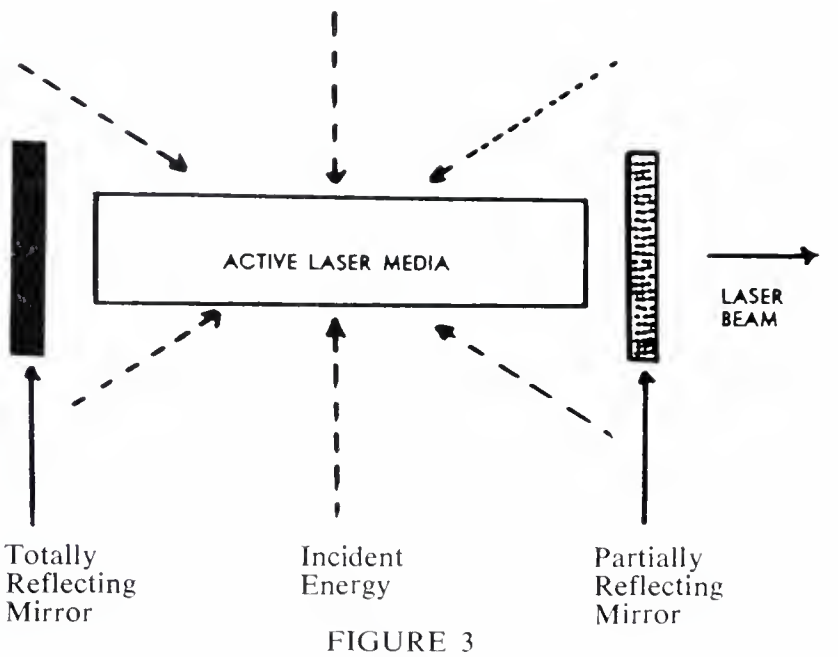


FIGURE 3

FABRY-PEROT INTERFEROMETER

The model I have used to date has been what is called the two level atomic system. In reality, most laser media have many more usable levels than two. The principles, however, in these media are the same as for those in the two level system. Figure 2 illustrates the three level system (in essence the ruby laser is an example of this type) and the four level system (an example of which is the neodymium laser). These examples show that the pumping energy (E_2–E_0 in the three level system and E_3–E_0 in the four level system) need not be the same frequency as the photon input and laser output frequency. Energy levels such as E_1 in the three level system and E_2 in the four level system are called meta-states, being between the ground and initial excited states. In the case of the ruby laser, the lifetime τ of an excited electron in E_2 of the three level system is in the range of nanoseconds. Then the electron decays to E_1 by a radiationless transfer of energy to the crystal lattice. This new level has a τ of three milliseconds and this value is adequate to sustain stimulated emission with the use of high intensity xenon flashlamps pumping the system. Four level systems give a greater population inversion than do two and three level systems for a given amount of pumping energy, and thus are more efficient. If it is imagined that there is another meta-state line in the three level system of Figure 2 where τ is such that a build-up of the excited population may occur there as well, then, should appropriate photons be available to introduce a second chain reaction, the laser will emit a light with two distinct frequencies. Of course, this argument can extend further to explain any number of distinct frequencies emitted in one laser beam.

There are a variety of methods for pumping a laser media which include: 1. optical pumping using high intensity light sources; 2. sparking an electrical current in an inert gas to ionize the molecules which are then inverted by collisions between the ions; 3. passing a current through a junction diode, causing inversion at the interface.

To finally become operable all laser systems have one final requirement, namely, a Fabry-Perot Interferometer (see Fig. 3). This consists of a pair of parallel mirrors placed at either end of whatever particular media is being lased. Although stimulated emission provides amplification by itself, the Fabry-Perot Interferometer reflects the amplified product back through the media to repeat the multiplicative process many times, the beam becoming even more intense. One mirror is always totally reflecting. The other mirror is always partially reflecting. This means a certain percentage of the light reaching it is reflected while the remaining amount is passed through. It is this "passed light" that is seen as the emitted laser beam. If the partially reflecting mirror is always in this state then a continuous laser beam is emitted providing the system continues to be adequately pumped and continuously supplied with appropriate photons for chain reaction. This type of device is called a continuous wave laser. Mechanical shutters must be used to control firing of the beam.

Should the appropriate photon input energy to fire the beam be different from the energy needed to pump the media (for the reasons outlined above), and you can supply the two independently of one another, then the beam fires only when a pulse of appropriate photon input is applied. These devices are called pulsed or burst lasers. A third type of laser, known as a Q-switched laser, has a partially reflecting mirror that is its own shutter[2]. The partially reflecting mirror is placed out of parallel while pumping occurs for a long period of time. Missing the normal stimulated emission losses caused by a functioning Fabry-Perot Interferometer, a very large population inversion builds up. Suddenly, the partially reflecting mirror is properly realigned for a selected time and a flood of atoms descend to the ground state releasing a very short but extremely powerful pulse of light.

The light that is ultimately emitted from any laser has four properties of importance that distinguish it from ordinary light. The first is its *monochromaticity*. This property follows from how the beam is created. All atomic changes of energy level release exactly the same quantity of energy (assuming that only two levels are involved) that must, from Planck's law, have the same frequency and (since $\lambda = \frac{c}{\nu}$, where ν is the frequency of the emitted radiation, λ its wavelength and c the speed of light) the same wavelength. The second property of importance is that laser light is light of a very high *intensity*. Thirdly, the light rays as they are emitted are at a high level of *collimation*, that is to say, very nearly parallel. Fourthly, the light has a high degree of *coherence*. Coherence has a precise mathematical definition, but broadly speaking, a beam of light is coherent if the waves that make it up have a consistent waveform in themselves over time and each has a consistent phase relationship with the other in space.

LASER MODELS USED IN OPHTHALMOLOGY

Even as recent as three to four years ago no one type of laser had been generally decided upon as being superior to the rest. Thus, laser systems were made using as the lasing media, ruby, YAG-neodymium (a yttrium-aluminum-garnett crystal doped with neodymium, the actual lasing substance) and argon. Today the argon laser has assumed the position of preeminence in ophthalmology and Figure 4 provides a very neat summary of the rationale for its use. When light of a single wavelength is passed through pigment epithelium and choroid, for example, the percentage that is absorbed can be measured. These absorption measurements can be made at many more single wavelengths and the results can be plotted as shown. From this curve, it can be seen that light with a wavelength of 400nM passes well through pigment epithelium and choroid while light of 600nM wavelength is nearly completely absorbed. Similar curves can be constructed, as shown, to represent blood vessels and media. Superimposed on these curves are the wavelength emission lines of various types of lasers that were developed into models useful in ophthalmology. To burn a structure in the eye, laser light must be absorbed by that structure. An ideal laser light would be completely transmitted by the media, leaving it intact, while being completely absorbed by pigment epithelium and choroid of blood vessels. This criterion narrows the field of choice down to the argon, YAG-neodymium or krypton lasers. However, a second important consideration in ophthalmology is power output, or alternatively, retinal power density expressed in watts/cm^2. As the power density delivered to the retina falls, both the *total* energy that must be delivered to the retina and the time of exposure necessary to produce a lesion must increase[3]. However, the eye is in constant, normal

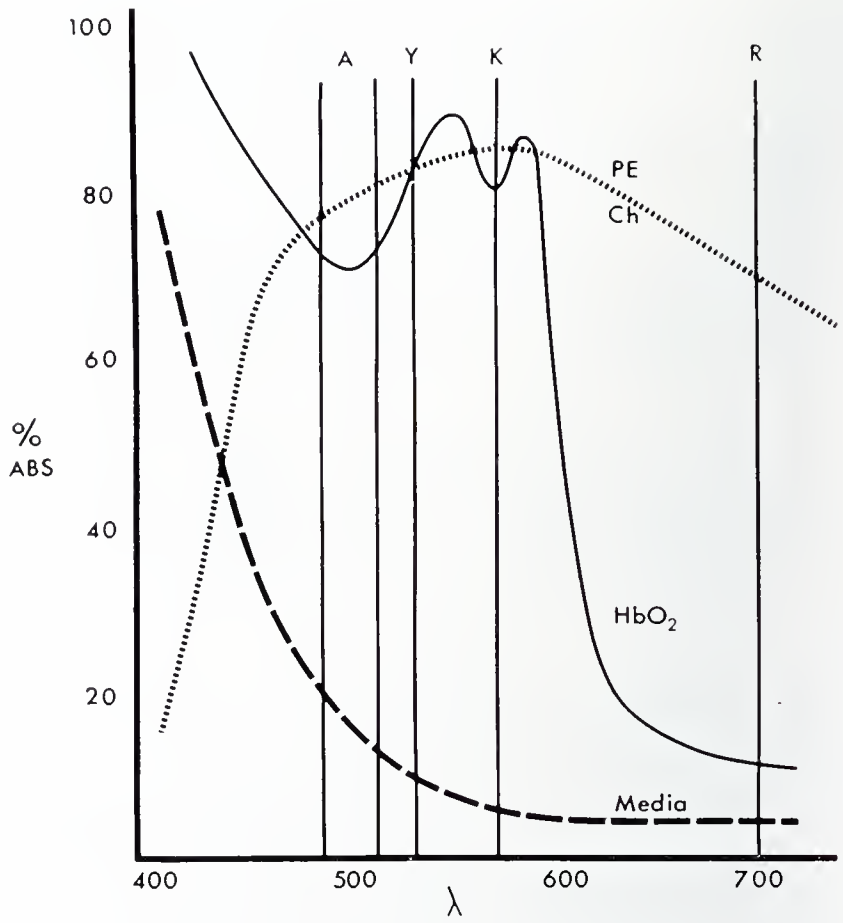


FIGURE 4

This graph indicates the amount of light absorbed by various structures in the eye as a function of the wavelength of the incident light. Superimposed on these curves are the major emission lines of various types of lasers. Note, for example, that from this graph it may be predicted that the ruby laser is a useful instrument for coagulating retina to choroid, but is not the best model to choose for photocoagulating blood vessels.

PE and Ch: pigment epithelium and choroid

HbO_2: oxygenated hemoglobin; thus this curve represents the absorption curve of retinal blood vessels

Media: those structures, including cornea and lens, through which the light must pass before it reaches the desired site of action

A: the argon laser with its two major emission lines at 488.0 and 515.5nM

Y: the YAG-neodymium laser with its major emission line at 530nM

K: the krypton laser with its major emission line at 568.2nM

R: the ruby laser with its major emission line at 694.3nM

physiological movement throughout the procedure. Consequently, one wishes to choose a laser with the smallest time of exposure necessary. Of the three mentioned, argon at the present time has this necessary high power output in a reliable, commercially available machine.

These argon models can be either continuous wave models or the newer burst or pulsed argon models. The continuous wave model is controlled by mechanical shutters, must be water cooled and requires special Hydro arrangements. It was the first argon type available, however, and thus this is the type of machine found in a major hospital like the Toronto General Hospital. More recent is the burst argon model. This

FIGURE 5

Close-up photograph of the control console shown in Figure 6. The desk on which the console sits is actually the laser itself and the beam is transported to the aiming device by means of the articulated metal arm shown emerging from the desk top in the upper left hand corner of the picture.

FIGURE 6

Photograph of the laser apparatus in use at the Toronto General Hospital. On the right hand side of the photograph is seen part of the desk that is the actual laser itself. On the desk is the control console and above that can be seen the articulated metal arm inside which the beam is travelling to the aiming device. The physician is shown adjusting the large contact lens placed in the patient's eye to keep the eye open during the procedure. His right hand controls the joy stick, which moves the entire apparatus not only from side to side, but forwards and backwards as well, bringing the parts of the patient's eye to be photocoagulated into view and into focus. The laser beam manipulator can also be clearly seen as the metal lever in the centre of the picture. This positions the aiming beam, visible as a green spot while focusing through the eyepieces, on the structure to be photocoagulated. The large knob below and to the right of the lever selects the desired spot size. The physician's right foot can be seen on the firing control switch which is pressed to fire the laser beam.

can be air cooled and is suitable for the individual ophthalmologist's office use. Figures 5 and 6 show various aspects of the continuous wave argon laser system at the Toronto General Hospital. The three basic laser variables that can be controlled by the physician are the power output, the time of exposure and the spot size. The smaller the spot size with a constant power output, the greater is the power density at the retina and the more severe the lesion created in the various retinal layers. Because the original beam is parallel, it can be focused accurately to extremely small spot sizes. The optics of the aiming device are such that the depth of eye that is in focus (whether it be the retina itself or blood vessels floating free in the vitreous in front of the retina) is automatically the area where the laser is focused to spot size. The regions in front of and behind the depth of focus should be spared serious injury because the beam is either still converging in the one case or diverging in the other. One way of putting this is "*What you see is what you hit.*"

THE CLINICAL PROCEDURE

Lasers at the present time have six actual and potential uses in medicine. These are: 1. welding retina to choroid; 2. occluding new vessels by coagulation; 3. forming iridectomies; 4. surgical incisions and cautery; 5. tumour necrosis; 6. holography of the fundus. Of these six, only the first two are in anything but the research phase of development. At the present time, the two major uses are spot-welding retina to

choroid and destroying neovascular growth. Neovascular growth may occur in a long list of diseases, but by far the most common is that growth occurring in diabetes[4]. In this disease, the new blood vessels float free in the vitreous and the laser is used to photocoagulate them before they become large or numerous. This type of management is only a treatment of symptoms and does nothing to prevent new growth occurring in the future. The postulated mechanism for all types of neovascularization is hypoxia causing the release of an extravascular intraretinal substance or factor that stimulates the new vessel formation. For this reason, laser photocoagulation may be combined with xenon-arc peripheral retinal destruction. The xenon-arc photocoagulator is an instrument that coagulates retinal tissue by an intense beam of white light from a xenon-arc lamp. It was used for coagulation many years before the laser but has largely been superceded by the laser because of the laser's superior focusing ability, much smaller spot sizes, shorter exposure times, and the fact that the xenon-arc with its white light emission covering the whole range of wavelengths damages to a greater extent eye structures otherwise unaffected by the laser. However, it is suitable for non-specific peripheral retinal destruction. This peripheral destruction, while not affecting visual acuity to a great extent, decreases oxygen demand of the retinal tissue resulting in less neovascular growth. Another condition that will cause retinal neovascularization is central retinal vein occlusion. Central retinal vein occlusion may happen in a variety of disease states, either commonly or uncommonly, and many diseases, including sickle-cell disease, malaria, polycythemia and dysproteinemia may have as a component of their problem retinal neovascularization.

The fusing of retina to choroid may be called for in three situations. The first of these is in the case of a retinal tear. The majority of retinal tears are of unknown etiology, although people with myopia seem more prone to develop a tear. Unless promptly attended to, vitreous seeps through into the tear and the sensory retina becomes completely detached from the pigment epithelium over an increasingly wide area. With what is now retinal detachment, comes increasing loss of visual acuity and degenerative changes in the sensory retina. The goal of laser photocoagulation is to place a ring of fused tissue around the lesion to stop any further tearing of the retina than has already occurred and to prevent this tear from progressing to total retinal separation. The second situation where retinal-choroidal fusion may be indicated is as a post-operative adjunct to surgery for retinal detachment. Should it be discovered post-operatively that the refusion of the retina to choroid has been incomplete and a small tear has been left unfused, laser photocoagulation may be employed to once again isolate this tear. In both these situations it is important to realize that successful laser fusion, like successful soldering, can only take place if the two elements to be fused, choroid and retina, are in contact. Once retinal detachment has occurred, the laser is useless and surgery must be performed. The third situation in which the laser can be used to unite choroid and retina is in that group of conditions known as macular vascular diseases. Once again, these diseases are of unknown etiology, but they do occur more often in the older age group. The first sign of disease is an area of retinal detachment just off the fovea caused by fluid leakage due to increased permeability of the choriocapillaris. The next stage of the disease is the presence of neovascularization in the area, finally progressing to hemorrhage. The exact area of choriocapillaris involved in the leakage may be well detected by fluorescein retinal angiography. If this area of leakage is then completely coagulated this progression is halted. Extreme care must be taken because of the beam's nearness to the fovea.

The laser operative procedure is very straightforward. The patient needs little preparation and no anesthetic. The appropriate pupil is dilated maximally before the procedure, usually with a few drops of 10% phenylephrine. The special contact lens to keep the patient's eye open is coated with 2% methylcellulose and inserted. The patient is asked to place his/her chin on the positioning chin rest provided and a strap is done up around the top of the head. The purpose of this is to try and keep the patient's head as still as possible. The room during the procedure is kept dark. The binocular aiming apparatus can control both the area of the eye viewed and the plane of focus. Within the field of view, the physician is free to move to any desired place, the green aiming spot a very weak laser beam projecting at all times into the patient's eye. Since the patient is in full control of his eye movements, he can be requested to look in any direction to help the physician visualize the desired intraocular area. The firing mechanism is activated by a foot control and when this is done there is a bright green flash in the field of view. The area that had been under the aiming spot can now be seen to be a small white circular patch in the surrounding red tissue. The aiming and firing is repeated with the variables of power, exposure time and spot size being adjusted appropriately in light of the appearance of the first lesion. This process is repeated until, as the case may be, the physician is satisfied he has photocoagulated the neovascular growth adequately, or until he has sealed in the area of retinal tear sufficiently. If the photocoagulation is done in the afternoon, the usual post-operative procedure is to have the patient spend that night in hospital wearing an eye patch, then the following morning for the ophthalmologist to check the results and, if all is well, the patient can then go home. The patient has his eyes rechecked at one week, four weeks and six months post-procedure by the ophthalmologist, who is looking to see how effective his treatment has been and for the presence of any new abnormalities. If the treatment was for neovascular-

ization, the photocoagulated vessels will gradually shrivel up and disappear from the vitreous and the ophthalmologist hopes no new abnormal vessels will grow to replace them.

Histologically, what happens when choroid and retina are coagulated? Of course, depending on the power setting, the area of retina that has absorbed laser light may be affected not at all, or so severely that the choriocapillaris is disrupted, with hemorrhage into the lesion. When speaking of lesions at clinically used settings, however, argon laser light causes selective coagulation of the outer retinal layers, principally the inner and outer nuclear layers and the pigment epithelium, while the inner retinal layers remain unchanged[5]. As well, the usual retinal detachment occurring between sensory retinal epithelium and pigment epithelium as an artifact of histologic sectioning is absent in an area that has been treated by laser photocoagulation. In these areas, the retinal epithelium and pigment epithelium remain firmly fused together even after sectioning.

The two immediate complications with the use of the laser are inadequate photocoagulation of a retinal vessel resulting in hemorrhage into the vitreous, and careless photocoagulation in the region of the fovea, destroying the patient's "hill of vision". Nothing can be done about the latter complication and the former one must be treated as soon as it is noticed during the procedure by adequately coagulating the vessel proximal to the hemorrhage. If hemorrhage is noted on one of the post-procedure checkups it is difficult to know whether to place the blame on inadequate previous treatment or on entirely new growth that has arisen since the last treatment. As with any vitreous hemorrhage, resolution will take place very slowly. Anywhere from a few days to months may be necessary for recovery, depending on the hemorrhage's initial size. As well, the vitreous may go on to residual fibrosis.

The three long term complications are failure of treatment, where a treated tear still goes on to retinal detachment, or new blood vessel growth which quickly replaces that coagulated, and what could be termed the multiplicative effect of treatment, where after initial photocoagulation has taken place neovacularization quickly starts again and this time is much more severe.

CONCLUSION

Medically related uses of the laser continue to be researched and developed. The four areas of most active research are the last four potential uses listed previously. Perhaps the most exciting is the research into developing holography as an ophthalmological tool. Three dimensional images of the interior of the eye, including the various layers of the retina, would be almost the ultimate in ophthalmoscopy.

In the meantime, lasers provide easy, accurate and safe access to structures not easily accessible surgically, for lesions that, once established, continue at the present time to be medically untreatable.

ACKNOWLEDGEMENTS

The author wishes to express his grateful appreciation for the help and advice of Dr. John Parker of the Department of Ophthalmology and Dr. Clive Mortimer of the Toronto General Hospital. Without them, I could never have completed this article. I also wish to express my appreciation to both of them for graphs and photographs so freely donated for use in this piece. Beyond the narrow confines of lasers, Dr. Parker must also be thanked for his willingness to satisfy my curiosity about optics in general. Easily some of my most pleasant "clinical teaching sessions" were the afternoons spent with Dr. Mortimer in the operating room. In both cases, it was all too easy to forget about my paper on lasers altogether.

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The 'Mustard' Report: The Future of Health Care in Ontario?

Terrence J. Colgan, 7T7

A recent Ontario Government report proposes large changes in the delivery of health care within this province. These proposals include recommendations that will tremendously affect medical students and doctors. This article reviews some of the relevant issues, for the prospective doctor, presented in the report.

In late 1976, the Ontario government passed legislation that made registration by all health professionals with their local Health District a mandatory prerequisite to registration under OHIP. Local Registration Certificates were issued to all physicians practicing prior to the bill, entitling them to OHIP billing.

In April the following year, Dr. Mary Brunner, upon nearing the completion of her residency as a Family Physician applied for registration as a Family Practitioner to her home region, the North Toronto Health District. Two months later, she received a refusal that subsequently denied her registration under OHIP. The reason given for this answer was that the present manpower needs within this area, as determined by the Ontario Provincial Government, had been successfully met the previous year.

The doctor was thus faced with two alternatives. She could choose a practice within her own chosen area without the coverage of OHIP and rely on patient-billing. Secondly, she could choose to move away from her home town to a recognized underserviced area where her practice would be entitled to OHIP privileges.

At the same time that Dr. Brunner read her refusal, a wife and husband were speeding toward St. Vincentville, a small Ontario town twenty miles from their farm. With them was their two year old daughter who was suffering from an acute asthmatic attack. An hour and a half later, the husband had returned to plowing the fields. The child, having been treated at the community clinic, was asleep in the house. Five years earlier a similar medical emergency may have cost a day's work during the critical planting season because of long travel time and hospital waiting.

Although these two incidents are obviously fictitious, the current 'Mustard' Report, even if adopted only in part, could result in similar events.

The 'Report of the Health Planning Task Force', or as it is more commonly labelled, the 'Mustard' Report, after the Chairman of the Committee, Dr. J. F. Mustard, was commissioned by the Ontario government to review the present health system and propose a viable, comprehensive scheme for the future[1]. The committee investigated virtually all aspects of health care delivery in Ontario – manpower, financing, institutions, research, education, and government organization.

Implementation of the report's recommendations is only vaguely discussed by the report. Consequently, the Health Ministry gave the 'Mustard' Report a cool reception. Nevertheless, the issues raised by this report are important to both the public and health professionals.

The 'Mustard' Report will be influential in determining the course of health care changes for all Ontario residents and medical personnel, including doctors. This review, however, will focus primarily on the impact of the 'Mustard' Report on the medical student and doctor. Since the report proposes large changes in the mode of practice, doctor placement, basic research, and medical education, these topics must be examined by every medical student. Implementation of the whole report would definitely frustrate many of the present expectations of both medical students and doctors.

However, it would be myopic and self-indulgent if the prospective doctor was not to recognize the aims of the report and health needs of Ontario while simultaneously realizing the report's potential effect on doctors.

The committee set out to "develop proposals for a comprehensive plan to meet the health needs of the people of Ontario"[2]. As indicated by the report findings, these health needs include improved hours of service, personnel distribution, quality of care, and service efficiency. The report's proposals were to design a system that would be able to provide for the "eventual coordination of health and social services"[3]. The third object of the committee was to suggest methods to slow or halt the rapidly rising health costs of the Health Ministry.

The recommendations begin by defining primary and secondary care levels and their respective responsibilities. The concepts of primary and secondary care sectors are complex and cannot be sufficiently explained within this review. However, primary care can very briefly be described as the level of basic patient-doctor contact in preventive care, diagnosis and treatment, and possible subsequent referral to the secondary care level. The primary level, according to the report, is to consist of various health professionals – nurses, physiotherapists, occupational therapists, psychologists, physicians, pharmacists, social workers, and other professionals as required by specific local needs.

The secondary sector receives patients only from the primary care level (incentive for this course is outlined in the report). This care level consists of "specialized

skills and faeilities not available in the primary eare seetor"[4]. The report emphasizes that the seeondary eare level must not be unique to hospital inpatient treatment. Rather, it must utilize ambulatory outpatient treatment in order to reduee eosts and inerease eonvenienee for the patient.

The framework of primary and seeondary care levels is far from gaining universal aeeeptanee as an eeonomieal and viable health system strueture. Indeed, the praetiee of sereening all patients through a primary level before reaehing a seeondary level may prove to be costly.

The report elaborates on this fundamental hcalth seheme. Under these basie preeepts, reeommendations inelude provisions for the administration of health delivery, researeh, and medieal edueation.

Government generally is renowned for its administrative eomplexity and often bewildering bureaueratic size. The 'Mustard' Report recommends an expansion of the Ontario government bureaueraey. Citing the urgent neeessity of overhauling the health system, the report exeludes the possibility of partieipation of regional government in the administration of health serviees[5]. Administration is to be eentralized in the Health Ministry.

The organization of health delivery within Ontario is to eonsist of a hierarehy of geographie units – with Queen's Park and the Health Minister at its apex. Ontario would be divided into administrative Regions, and these Regions subsequently subdivided into Distriets. All Distriet Couneils "should eonsist of ten impartial members appointed by the Lieutenant-Governor in Council on the adviee of the Minister of Health"[6].

Critieism of the 'Mustard' Report must be levelled at the proposed Health Ministry organization. The proposed Distriet Couneils are to be appointed; this method of seleetion deserves a eloser serutiny. In the opinion of this author, eleetion of members is preferable. Seeondly, the proposed loeal health areas (distriets and regions) are not eoineident with the regional governments in Ontario. In the future, this incongruity may prevent direet eleetion of Distriet Health Couneils using the existing politieal boundaries.

Sinee the Distriet Health Council is not to be reimbursed finaneially, the eouncil's impartiality is not direetly endangered by members' peeuniary self-interest. However, sinee District Health Couneils have extensive powers over health care and personnel, the implieations of an *appointed* body still have profound effects on the medical doetor. Despite the report emphasizing that members are to be 'impartial', the reeent history of the provineial government provides the realization that government patronage and nepotism are hardly unknown within the provineial administration. Indeed, any organization using appointment as its sole selection of membership is liable to descend into the depths of inefficieney, self-interest, and corruption. Fortunately, Ontario has seen the abuse of public money and power only to a limited extent, without severe mismanagement and ineptitude. An appointed District Council exposes the provincial health system, including doetors, to these dangers.

In addition, appointed bodies are not direetly responsible to the publie, but are responsible to the Ministry alone. Government appointees ean be insensitive to loeal demands without the election 'threat' whieh can insure eooperation and response. Appointed bodies, unlike eleeted assemblies, do not have to respond to publie serutiny with honesty and with information. The reeent investigation into the Ontario Hydro dealings have illustrated the abuse of publie trust and money that may follow deeisions by appointed eommittees.

The second eritieism of the proposed organization eoneerns the boundaries of the Health Distriets. The report insists that the urgeney of health problems overrides the judieious use of existing loeal governments. This impetuosity has resulted in the reeommendation that yet another level of administration be established in Canada. In a eountry that is already laden with disputes and inaetion due to disagreement between levels of government, the preseription of a vital health service organization that does not eoineide with any of the other three existing levels ean be interpreted as a ease of national masochism. This problem should not be viewed by medieal students as a purely aeademie issue. If these proposals are adopted this organization could make sueeessful health management difficult or impossible.

Having established the organization of the proposed Health Ministry, some of the aspects of it ean be examined.

> *Recommendation #10:*
>
> *Manpower planning and control should be introduced by having each District Health Council decide, within provincial guidelines, the number of positions to be established for various categories of health personnel, according to district requirements. A physician holding one of these positions would be eligible for registration under the Ontario Health Insurance Plan. Individual physicians would be free to seek out the positions that they wished to fill in all regions of Ontario*[7].

Although the report is vague on the specific committee that is to judge a doctor's applieation to practiee in an area, the Area Health Serviees Management Board is the implementation body, and ean be assumed to be the aetual deeisive body in this affair[8].

This reeommendation is probably the item of eonsequenee to most medieal students. Provincial need to reduee the over-use of medical serviees in urban areas

and increase the supply of health personnel to underserviced areas could result in the establishment of appropriate district manpower limits. Any physician who applied to a District in which there were no vacancies would be left with the choice of private practice (not insured by government), or selection of another district.

The tradition of the physician's freedom of movement and selection of geographic locale would obviously be curtailed. However, the report also suggests that no physicians presently practicing within any district be required to change location. All these physicians will receive registration under OHIP. The significance of this "grandfather clause" is immense for the medical student.

The current medical graduates of Ontario together with immigrant doctors may well bear the full brunt of the Ontario government and people, just demanding correction of the maldistribution of medical doctors. This conclusion is not a pleasant prospect to those medical students who wish to practice in the more crowded urban areas.

The implications of this proposal regarding further education and teaching are also not clear. If a medical graduate had a desire to practice while maintaining some part-time teaching or research that could only be performed within the city, would this desire be feasible if that city had its quota of doctors? What effect does this proposal have on the practicing physician who is contemplating specialization and a return to the city?

In conjunction with these questions, doubts arise as to the effectiveness of this proposal in delivering high quality health care. The 'Mustard' Report foresees the development of a private, non-OHIP practice alongside the OHIP practice[9]. Thus, the public would have two alternatives in selecting a private physician. The provision for non-insured private practice may be a dangerous alternative to OHIP. This policy may encourage distinctions between a 'rich-man's' doctor and a 'poor-man's' doctor. The British health scheme presently finds coping with this dualism a difficult and sensitive issue. Would such a discrepancy in payment also be reflected in a discrepancy in the quality of health care?

With these manpower policies in mind, the doctor or medical student can appreciate that the District Health Council may one day become a great influence on his/her profession. Not only does the District Health Council establish policies of manpower needs, it also is responsible for policies of coordination of sectors, services, facilities, and information within the district.

Despite the report's claim that it is decentralizing delivery and administration of health services, the actual proposals seem to indicate the contrary[10]. All levels of the health administration are responsible not to the local government but to the central Ontario legislature[11]. Financial control is delegated not to the District Health Council, but to the provincial level[12]. Lastly, the planning of the District Health Council is to be strictly within provincial guidelines[13]. Therefore, doctors should note that many important decisions will be made by the central authority, the provincial government, not the local District.

Despite its length, a mere seventy-seven pages, the report is concise and contains many details that cannot be covered in this review. However, some recommendations merit mention because of their influence on doctors.

"Separated, isolated functioning should be discouraged", declares the 'Mustard' Report[14]. Group practice and clinics are to be the functioning units of both primary and secondary care. This recommendation is not surprising considering the increasing voluntary group practice units that have evolved not only across Ontario but throughout Canada and the world. Still, it may frustrate some medical doctors with a desire for independent practice. This recommendation of the report was prompted by the need for twenty-four hour, seven days a week health service in all levels of health care.

For those students or doctors considering basic research, the future is indeed bleak; basic research expenditures are to be cut from .82% of the Health Care Expenditure to .75%. Applied and Developmental Research is to be tripled from its present level of .82%[15]. The report does not venture forth into the quagmire of the distinction between basic and applied research. Instead, it blandly assumes that the distinction is not only real, but can be discerned when issuing grants. Unfortunately, the applicability of the product of research is often not known until after the research has been completed. In addition, the report ignores the possibility that Ontario, being one of the richest areas in the world, may have a moral responsibility to maintain basic research.

Review of medical competence would be increased under the proposals. The College of Physicians and Surgeons would witness much more activity on the part of the Ministry in this area. Peer review within the primary and secondary care sectors of the District (a "health audit") would be routine. In addition, the report suggests that the performance of a physician in practice would be reviewed by the College periodically.

This article has focussed on the impact of the 'Mustard' Report on the student and doctor, and has only touched on the report's consequences for the public. A thorough examination of the report must consider these topics as well. Neither of the initial anecdotes about doctor or farmer, when taken singly, can adequately illustrate the issues involved in health delivery changes. Rather, an integrative approach is necessary; one that is mindful of both public and professional needs.

Searching for alternative solutions to proposals of the 'Mustard' Report is a difficult task. Yet, this course is necessary if medical students are to shoulder their responsibility for health care. This situation is one where doctors and students must react with imagination and vigour to both social and political demands to preserve and create their own desired lifestyle while providing adequate medical care for the future population of Ontario.

FOOTNOTES

1. Available at the Ontario Government Bookstore, Bay and College, Toronto.
2. 'Report of the Health Planning Task Force'. Ontario Government Publication, 1974. p. vii.
3. *Ibid.* p. vii.
4. *Ibid.* p. 15.
5. *Ibid.* p. 31.
6. *Ibid.* p. 28.
7. *Ibid.* p. xii.
8. The District Health Council forms general policy for the District at its meetings. The Area Health Services Management Board is the 'workhorse' of District management. This Board is constituted of full-time, paid administrators and staff, and is concerned with policy implementation.
9. 'Report of the Health Planning Task Force'. p. 66.
10. *Ibid.* p. 8.
11. *Ibid.* p. 24.
12. *Ibid.* p. 27.
13. *Ibid.* p. 34.
14. *Ibid.* p. 7.
15. *Ibid.* p. 72.

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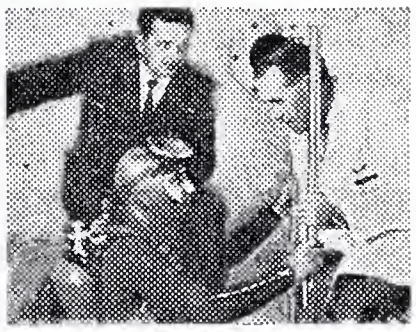

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